JOURNAL

OF THE

American Veterinary Medical Association

FORMERLY AMERICAN VETERINARY REVIEW

(Original Official Organ U. S. Vet. Med. Ass'n.)

H. Preston Hoskins, Secretary-Editor, 537 Book Building, Detroit, Mich.

Vol. LXXVII, N. S. Vol. 30 September, 1930 No. 3

AN ILL-ADVISED PARAGRAPH

Quite a number of our members have written in for the purpose of directing attention to an article that appeard in the July issue of the *Country Gentleman*. This article described, in a popular way, the development of hemorrhagic septicemia aggressin, by Dr. Wm. S. Gochenour, of the U. S. Bureau of Animal Industry. On the whole, it was the sort of an article that directs favorable attention to the veterinary profession in general, and to the value of veterinary research in particular. However, in the opinion of practically all veterinarians who read and commented on the article, its value was almost completely spoiled by the concluding paragraph, which read as follows:

And not only is the remedy cheap, but it has the additional advantage of not needing the services of a veterinarian for administration. The county agent can do the job, or instruct the farmer in the simple technic of doing it himself.

In cases of this kind it is a rather difficult matter to place the blame for such a gross misstatement of fact. Our first impulse naturally would be to blame the editor of the *Country Gentleman* for allowing such an incorrect statement to appear in print. He would probably be inclined to "pass the buck" to the author of the article, who is supposed to be a pretty well informed writer on subjects agricultural. The author, in turn, would probably

pass the blame on to the "source" of his information, whoever or wherever that might be.

Previous experiences with similar cases have served only to demonstrate how unsatisfactory it is to secure correction of such misstatements. Unfortunately the damage is done when the misstatement first makes its appearance in print. Retractions made subsequently are very likely to be overlooked and not connected with the original statement, and sometimes they lead to endless acrimonious discussions which do nobody any good.

Furthermore, we are not inclined to be unduly harsh in criticising those responsible for errors of this kind. Almost invariably they are unintentional, and we are rather inclined to the more charitable view that the favorable publicity given the veterinary profession by most farm papers and publicists in general outweighs the occasional lapse made by an agricultural editor or the author of an article whose intentions are perfectly good.

Might we not take a little of the blame unto ourselves? Is there not more or less of an indictment against the veterinary profession for allowing even an impression to become current that a county agent or a farmer can use such a delicate and potentially dangerous biological product as hemorrhagic septicemia aggressin?

Any aggressin is a two-edged sword and calls for the utmost in careful discrimination, professional judgment and technical skill in its administration, if satisfactory results are desired. Aggressins are second only to living viruses, from the standpoint of the damage that may be done through their improper use.

MORE PUBLICITY

The June issue of the *American Druggist* contained an article entitled "Veterinary Science," by T. Swann Harding, which told "how druggists may help farmers in the cure and preservation of animals," according to the subtitle. The opening paragraph of the article is one calculated to get a rise out of most physicians if not a few veterinarians. We quote it:

> Not many years ago the term "horse-doctor" was one of derision, if not of reproach. It still draws a laugh among ill-informed laymen, and physicians who should know better still tend to rate a veterinarian somewhere between a chiropodist and a chiropractor. But, as Will Rogers says, "He has to know something, because he can't ask a horse where it hurts," and the United States Bureau of Animal Industry has, during recent years, been largely instrumental in so raising the quality of our veterinarians and coordinating their work that today they are far better organized to combat animal disease than are human physicians to eradicate human disease.

The article is a rather lengthy one and, in a general way, is an account of the activities of the U. S. Bureau of Animal Industry. The subtitle above referred to would be less misleading if it read: "How Uncle Sam is helping farmers to cure and prevent animal diseases." With the exception of several prescriptions for demodectic mange in dogs, there is little information given the druggist to pass on the farmer that is not available to the latter through more direct channels. On the other hand, the information given the druggist, for his own enlightenment, concerning modern veterinary activities, makes the article well worth while.

APPLICATIONS FOR MEMBERSHIP

(See July, 1930, JOURNAL)

FIRST LISTING

ADAMSON, CECIL T. Dept. of Agriculture, State House, Des Moines, Iowa.
D. V. M., Iowa State College, 1929
Vouchers: C. R. Fry and J. A. Barger.

ALEXANDER, RAYMOND HILTON Box 403, Harrisburg, Pa.
D. V. M., Kansas State Agricultural College, 1930
Vouchers: Joseph P. Scott and H. F. Lienhardt.

CALLAHAN, FRANK HOWARD Shelton, Nebr.
D. V. M., Kansas State Agricultural College, 1929
Vouchers: E. J. Frick and E. R. Frank.

CUTHBERT, ROBERT GILBERT 1663 W. 4th Ave., Vancouver, Brit. Col.
B. V. Sc., Ontario Veterinary College, 1923
Vouchers: W. H. McKenzie and J. G. Jervis.

EICHELBERGER, ALEXIS MARTIN 264 S. Crescent Drive, Beverly Hills, Calif.
D. V. M., Cincinnati Veterinary College, 1909
Vouchers: L. M. Hurt and L. F. Conti.

GIBBS, CHARLES SHELBY 9 Phillips St., Amherst, Mass.
D. V. M., Michigan State College, 1930
Vouchers: John B. Lentz and Henry Van Roekel.

GOCHNAUER, OSCAR BRUCE 2811 Garfield St. N. E., Minneapolis, Minn.
D. V. M., Ohio State University, 1930
Vouchers: H. C. H. Kernkamp and W. L. Nilson.

HARDY, LEO VANTUYLE 562 N. Lexington Ave., Saint Paul, Minn.
D. V. M., St. Joseph Veterinary College, 1920
Vouchers: G. E. Totten and M. E. Schwab.

HENTHORN, HAROLD ROY 511 Douglas St., Sioux City, Iowa.
D. V. M., Kansas City Veterinary College, 1918
Vouchers: Frank Breed and C. H. Covault.

HIGGINS, JOHN MERRITT 1528 E. 6th St., Tulsa, Okla.
D. V. M., Cincinnati Veterinary College, 1915
Vouchers: C. R. Walter and O. E. Robinson.

HULL, MARTIN 1112 S. Broadway, Santa Ana, Calif.
D. V. M., San Francisco Veterinary College, 1913
Vouchers: Fred C. Wright and J. H. Pullin.

IKARD, WM. LEWIS Box 301, Jerome, Idaho.
D. V. M., Kansas State Agricultural College, 1921
Vouchers: W. A. Sullivan and A. J. Dickman.

JOHNSTON, CARL A. Box 415, Lewiston, Idaho.
V. S., Ohio State University, 1904
Vouchers: J. R. Fuller and W. A. Sullivan.

KENDRICK, ARCHIE DAVIS — Homer, La.
D. V. M., Kansas City Veterinary College, 1914
Vouchers: J. Arthur Goodwin and C. M. Heflin.

MEIER, RAY — Smithville, Mo.
D. V. M., St. Joseph Veterinary College, 1918
Vouchers: J. D. Ray and J. L. Jones.

MILTON, MORLEY H. — Chilliwock, Brit. Col.
B. V. Sc., Ontario Veterinary College, 1911
Vouchers: T. H. Jagger and J. G. Jervis.

MORROW, JOHN KENNETH — Box 576, Huntington, L. I., N. Y.
D. V. M., Ohio State University, 1930
Vouchers: W. F. Guard and Walter R. Krill.

PARRY, RICHARD M. — 110 N. Dorcas St., Lewistown, Pa.
V. M. D., University of Pennsylvania, 1930
Vouchers: Vincent C. Moyer and E. P. Althouse.

QUILTY, MARK A. — Box 757, Lake Charles, La.
D. V. S., University Veterinary College of Kansas City, Mo., 1903
Vouchers: J. Arthur Goodwin and E. Pegram Flower.

SCHULZ, CARL A. — 123 E. Lexington, Independence, Mo.
D. V. S., St. Joseph Veterinary College, 1909
Vouchers: J. D. Ray and J. L. Jones.

SLATTER, ERVIN EDWIN — 50 E. Buchtel Ave., Akron, Ohio.
D. V. M., Ohio State University, 1930
Vouchers: W. F. Guard and Walter R. Krill.

STOVER, DONALD EUGENE — 1738 Lewis Ave., Long Beach, Calif.
D. V. M., State College of Washington, 1930
Vouchers: W. L. Curtis and A. G. Feers.

SUNDSTROM, CARL CLARENCE — 2121 Pico Blvd., Santa Monica, Calif.
D. V. M., Colorado Agricultural College, 1930
Vouchers: W. L. Curtis and L. E. Pike.

THIBEAULT, CORNELIUS — 80 Lowell St., Reading, Mass.
D. V. M., Ohio State University, 1929
Vouchers: Wm. M. Evans and H. W. Jakeman.

TULLY, HOMER GILBERT — 1320½ N. Harper Ave., Hollywood, Calif.
D. V. M., Cornell University, 1929
Vouchers: L. E. Pike and L. M. Hurt.

WARN, CHARLES C. — 836 W. Slauson Ave., Los Angeles, Calif.
M. D. V., McKillip Veterinary College, 1911
Vouchers: A. T. Ferguson and W. L. Curtis.

WILSON, JAMES EWELL — 2121 Pico Blvd., Santa Monica, Calif.
D. V. M., Colorado Agricultural College, 1930
Vouchers: W. L. Curtis and L. E. Pike.

WIMER, HARRY EDWIN — Eagle, Idaho.
D. V. S., Kansas City Veterinary College, 1911
Vouchers: W. A. Sullivan and A. J. Creely.

YOUNG, EDWARD LEE — Grandview, Mo.
D. V. M., Kansas City Veterinary College, 1912
Vouchers: J. D. Ray and J. L. Jones.

Applications Pending

SECOND LISTING

Argue, Cameron W., c/o A. S. P. C. A., 24th St. & Ave. A, New York, N. Y.
Bradshaw, Geo. B., Box 725, Jackson, Miss.
Delaplane, John P., Ohio State Univ., Columbus, Ohio.
Elliot, John Taylor, R. R. 2, Uxbridge, Ont.
Evers, John R., Bristol, Wis.
Fidler, Charles Edmond, 826 N. 5th Ave., Kankakee, Ill.
Halpin, Neil J. C., 80 N. Daisy Ave., Pasadena, Calif.

Holmes, Herbert Nash, Buffalo, Ill.
Hostetler, Orval Herbert, Box 133, Nampa, Idaho.
Hummon, Ormond J., Leipsic, Ohio.
Jones, Thomas Benton, 2142 W. Van Buren St., Phoenix, Ariz.
Jorgensen, A. B., Antigo, Wis.
Krieger, Gustave Louis, Box 1015, Bartlesville, Okla.
McCrory, Bryce R., State Laboratories, Reynoldsburg, Ohio.
Mohri, V. C., Lt. Ralph W., 324 Garland Ave., Takoma Park, Md.
Moore, Kenneth Russell, Woodville, Ont.
Randall, Glen H., 1855 N. 26th St., Kansas City, Kans.
Richards, W. L., Morrisonville, Wis.
Rosengren, M. W., Ackley, Iowa.
Schantz, Lansing C., Constableville, N. Y.
Thom, Myron A., 816 S. San Pedro St., Los Angeles, Calif.
Tripp, Lynn Howard, Rm. 93, County Court House, Albany, N. Y.
Zingg, Henry F., New Glarus, Wis.

The amount which should accompany an application filed this month is $6.67, which covers membership fee and dues to January 1, 1931, including subscription to the JOURNAL.

COMING VETERINARY MEETINGS

Tulsa County Veterinary Association. Tulsa, Okla. September 11, 1930. Dr. J. M. Higgins, Secretary, 1528 E. 6th St., Tulsa, Okla.

New York State Veterinary Medical Society. Ithaca, N. Y. September 15-16, 1930. Dr. C. E. Hayden, Secretary, 110 Irving Place, Ithaca, N. Y.

Georgia State Veterinary Medical Association. Columbus, Ga. September 15-16, 1930. Dr. J. E. Severin, Secretary, 1039 Marietta St., Atlanta, Ga.

Southeast Georgia Veterinary Association. Columbus, Ga. September 15-16, 1930. Dr. Hugh F. J. Arundel, Secretary, Statesboro, Ga.

Eastern Iowa Veterinary Association. Hotel Montrose, Cedar Rapids, Iowa. October 7-8, 1930. Dr. Paul V. Neuzil, Secretary, Blairstown, Iowa.

Maine Veterinary Medical Association. Bangor, Me. October 8, 1930. Dr. L. E. Maddocks, Secretary, R. F. D. 2, Augusta, Me.

Kansas State Agricultural College Conference for Veterinarians. Kansas State Agricultural College, Manhattan, Kans. October 16-17, 1930. Dr. R. R. Dykstra, Dean, Kansas State Agricultural College, Manhattan, Kans.

International Association of Dairy and Milk Inspectors. Hotel Statler, Cleveland, Ohio. October 22-24, 1930. Dr. Paul P. Brooks, Secretary, State Department of Health, Albany, N. Y.

THE TRANSMISSION OF PULLORUM DISEASE AMONG SEXUALLY MATURE FOWLS*

By H. C. H. KERNKAMP,† *St. Paul, Minnesota*

Division of Veterinary Medicine, University of Minnesota

The widespread distribution and increasing prevalence of pullorum disease (bacillary white diarrhea) makes it one of the most important diseases of the domestic fowl (*Gallus domesticus*). In 1909, Rettger and Stoneburn[1] discovered that the causative organism of pullorum disease often is found in eggs laid by affected hens. This led to further researches which pertained to the transmission of the infecting agent to the embryo and newborn through the medium of the egg. The results of these studies pointed very clearly to the fact that chicks often harbor the infection at birth. These findings have been confirmed by many other investigators. The transmission of pullorum disease through the medium of the egg, from the adult to the offspring, is a basic factor in connection with present-day methods of control. The transmission of the disease among sexually mature fowls has received much less attention and is not generally considered to be of great import. In fact, conflicting views exist as to whether contact infection actually occurs among sexually mature fowls. These views are in some cases supported by experimental data. It was with the hope of obtaining additional data on this phase of the pullorum disease problem that the following studies on the transmission and spread of the disease among sexually mature fowls were undertaken.

HISTORICAL

Rettgér, Kirkpatrick and Stoneburn,[2] in 1912, were the first to investigate the problem of the transmission of pullorum disease among sexually mature fowls. Their studies indicated quite conclusively that cohabitation is a factor in the spread and transmission of pullorum disease from infected to non-infected mature fowls. It was assumed that the food and litter were contaminated by the organisms and that the infection gained entrance into the body *per os*. This channel of infection was later considered not to be of primary importance. In fact,

*Published with the approval of the Director as Paper No. 942 of the Journal Series of the Minnesota Agricultural Experiment Station. Received for publication, Mar. 26, 1930.

†R. E. Lubbehusen formerly a member of the Staff of the Division of Veterinary Medicine, University Farm, aided in securing some of the material here reported.

according to Rettger, Kirkpatrick and Card,[3] rarely if ever did the transmission of the disease occur in this manner. They reached this conclusion after conducting several experiments in which infected (artificially) and non-infected birds were confined in the same pen for periods of from five to seven months. The non-infected birds at the end of this time failed to show evidence of infection. In another pen, where infected and non-infected hens were being confined, a male bird was introduced. It is reported that subsequent to the introduction of the male, one of the non-infected hens gave evidence of infection according to the agglutination test. The male was also positive serologically at one of the test periods. This led to the opinion that the male bird might serve as a passive transmitter of the infection by coitus. This idea has received further support from some of the studies reported by Brunett.[4]

Brunett placed 17 hens which were negative to the agglutination test for pullorum disease in a pen with 22 hens which were positive to the same test. For seven months these two groups of hens were in direct contact. During this period none of the non-reacting hens ever showed evidence of infection as indicated by the agglutination test. After the seventh month, three non-reacting males were placed with the females. Four months later, two of the original non-reacting hens showed positive reactions. After another four months, or eight months from the time the males were first placed in the pen, three more of the original non-reacting hens showed positive reactions. Agglutination, however, occurred in a very low dilution (1:10). Eight of the original 17 non-reacting hens were still alive 20 months after the male birds were first placed in the pen. They were then destroyed and carefully examined bacteriologically. Four showed definite evidence of being infected. It was assumed that the infection had not been transmitted to the non-reactors until the male birds had been introduced into the pen.

Luttschwager[5] emphasizes that when birds are kept in small enclosures, the chances for pullorum disease spreading through the medium of infected feces is at its best. He notes further that the male bird is not a factor in the spread of the disease among adult fowls.

Infected and non-infected fowls were penned together for a period of twelve months by Edwards and Hull.[6] The non-infected hens were negative to the agglutination test for pullorum disease when they were placed with the infected ones. They had been

tested twice previously, at intervals of six months, and each time the test showed them to be negative. After the birds had been together for three months, one of the negative hens gave a positive reaction to the agglutination test. A second hen reacted for the first time seven months from the beginning of the contact period. After eight months of contact with positive-reacting hens, two more of the original non-reactors showed positive agglutination. The fifth bird to change from a negative to a positive reaction occurred ten months from the beginning of the contact period. Twelve months from the beginning of the experiment, the birds were destroyed and all of the non-reactors, which later showed positive reactions after association with infected hens, yielded *S. pullorum* on culture. Cultures made from those which remained negative to the test throughout the contact period were sterile. No male birds were included in this experiment.

Contrary findings are reported by Doyle.[7] He reports an experiment in which he studied the factor of contact infection among adult fowls. Fifty naturally infected birds were housed and penned with 30 healthy birds for a period of one year. During this time agglutination tests were made on the blood of these birds at regular monthly intervals, with the result that at no time did any of the healthy fowls show evidence of infection serologically. Doyle concluded that contact infection was not an important factor in the spread of pullorum disease among sexually mature fowls. The report did not make clear whether or not male birds were kept with the flock during the period of the experiment.

Experimental

The object of the experiment was to determine whether or not pullorum disease is transmitted by contact between infected and non-infected sexually mature fowls. The agglutination test (tube test) served as the basis of selection between infected and non-infected birds.

The birds were housed in a well-lighted and ventilated enclosure and had the privilege of an out-of-door pen or runway. The house or sheltered enclosure provided 360 square feet of floor space. The floor was of cement and at all times was covered with straw litter. Open water-vessels and feed-hoppers were located in the house as were also the roosts and trap-nests. The out-of-door pen provided 648 square feet of ground space. The

ground was devoid of all vegetation. The birds were permitted the runway in favorable weather only. The straw and litter was removed and the floor swept clean about once in every six weeks. The dropping-boards were cleaned every second day.

The investigation comprises two experiments or parts. In general, the second experiment is a duplication of the first but was conducted at a later date. However, because there were a few rather significant and important differences in the way certain parts of the experiment were conducted, we prefer to treat them as experiments I and II.

Experiment I

This experiment was begun in November and December of 1927 and continued until January, 1929. Fifty-eight fowls were used. Of this number 29 were hens which were positive to the agglutination test for pullorum disease; 2 were hens that were classed as doubtful or suspicious reactors; 24 were hens which were negative to the agglutination test and 3 were roosters which were also negative serologically. The negative birds were obtained from flocks in which more than 96 per cent of the birds showed no reaction to the agglutination test and in which little or no trouble from bacillary white diarrhea was reported. Each bird was tested at the time it was placed in the experiment pen and it was planned that each should be tested at regular monthly intervals throughout the period of the experiment. At the termination of the experiment all surviving birds were to be destroyed and examined bacteriologically and for any gross evidence of the disease.

Commencing in November, an agglutination test was run on each bird once a month for five months, in some cases, and four months in others. This period was followed by a four-month interval, during which no tests were made on any of the birds. The "no-test" period was occasioned by circumstances over which we had no control and while it is extremely unfortunate, the results would not be particularly affected. However, beginning again at this point, nine months from the start of the experiment, regular monthly tests were conducted until the termination of the investigations with this group of fowls. Table I shows the results of the periodic testing.

The tabulated data show that of the 29 birds which reacted when tested by the agglutination test at the start of the experiment, 21 showed gross lesions of pullorum disease when destroyed

TABLE I—*Results of the serologic diagnoses, bacteriological examinations for S. pullorum and observations for gross lesions characteristic of pullorum disease in the birds of experiment I*

BIRD	SEX	RESULTS OF SEROLOGIC EXAMINATIONS										RESULTS OF BACTERIOLOGICAL EXAMINATIONS	RESULTS OF GROSS PATHOLOGICAL EXAMINATIONS
		NOV.	DEC.	JAN.	FEB.	MAR.	AUG.	SEPT.	OCT.	NOV.	DEC.		
P20	F	+	+	+		+	+	+	+	+	+	+	+
53	F	+	+	+	+	+	+	+				+	+
R113	F	+	+	+		+	+	+	+	+		+	+
151	F	+	+	+		+	—					+	+
R164	F	+	+	+		+	+	+				+	+
202	F	+	+	+	+	+	+	+				+	+
215	F	+	+	+	s	+	—	+	+	+		+	+
240	F		+	+	+	+	+	+	+	—		+	+
241	F	+	+	+	—	+	+	+	+	+		+	+
242	F	+	+	+	—	+						+	+
248	F		+	+	s	+	+	+	+			+	+
279	F		+	+	+	+	+	+	+			+	+
285	F		+	+	+	+	+	+	+			+	+
307	F		+	+	—	+	+	+	+	+		+	+
315	F		s	+	s	+	+	—	+			—	—
316	F		+	+	+	+	+	+	+	+		+	+
321	F	+	+	+		+	+	+				+	+
327	F	+	+	+	s	+	+	—				—	—
342	F	s	+	+	s		+	—	+	+		+	+
344	F	+	+	+	+	+	+					—	—
346	F	+	+	+	+	—	+	+	+	+	+	+	+
366	F	+	+	s	+	+	+	+				—	+
405	F	+	s	+	+	+	+	+				—	+
450	F	+	+	+	—	+	+	+	+	+		—	—
489	F	+	+	+	+	+	+	+				—	+
503	F		+	+	+	+	+					—	+
509	F		+	—	+	+	+	+	+	+	+	+	+
559	F	+	+	+	+	+	+					+	+
575	F		+	+	+	+	+	+				+	+
825	F		+	+		+	+	+				+	+
842	F	+		+	—	+	+	+	+	+		—	—
34	M	—	—	—	—		—	—	—	—		—	—
100	F		—	—	—	—						—	—
533	F		—	—	—	—	—	—				—	—
537	F	—	—	—	—	—	—					—	+
551	F	—	—	—	—	—	—					—	—
552	F	—	—	—	—	—	—					—	—
585	F		—	—	—	—						—	—
590	F		—	—	—	—						—	—
593	F	—	—	—	—	—						—	—
819	F		—	—	—	—	—	—				—	—
824	F		—	—	—	—	—	—	—	—		—	—
829	F		—	—	—	—						—	—
837	F	—	—	—	—	—						—	—
839	F		—	—	—	—	—	—				—	+
F209	F	—	—	—	—	—	—	+				—	—
251	F		—	—	—	—	—	—	+	+	—	—	—
268	F		—	—	—	—	+	—	+			+	—
302	F		—	—	—	—	—	—	+			—	—
362	F	—	—	—	—	—	+	—	+	—		—	—
483	F	—	—	—	—	—	+	+	+	+	+	+	+
584	F	—	—	+		+						+	

TABLE I—*Continued*

BIRD	SEX	RESULTS OF SEROLOGIC EXAMINATIONS										RESULTS OF BACTERIOLOGICAL EXAMINATIONS	RESULTS OF GROSS PATHOLOGICAL EXAMINATIONS
		NOV.	DEC.	JAN.	FEB.	MAR.	AUG.	SEPT.	OCT.	NOV.	DEC.		
591	F	—	—	—	—	—	+	+				—	—
835	F		—	—	—	—	+	+	+			+	+
838	F		—	—	—	+	+	+				+	+
1184	F		—	—	—	—	+	+		+	+	+	+
P	M	—	—	—	—	—	—	—	—	—	—	—	—
Y	M		—	—	—	—	—		—	—	—	—	—

F = female, M = male.
— = no agglutination.
s = agglutination at 1:25 and no agglutination at 1:50 and 1:100.
+ = agglutination at 1:25 and 1:50 and either agglutination or no agglutination at 1:100.
+ in bacteriological column = *S. pullorum* isolated.
— in bacteriological column = *S. pullorum* not isolated.
+ in pathological column = gross lesions characteristic of pullorum disease found at autopsy.
— in pathological column = gross lesions characteristic of pullorum disease not found at autopsy.

and from each *Salmonella pullorum* was isolated. The agglutination records of eleven of the 21 birds show they were positive each time the test was conducted. Four represent the lowest number of consecutive tests and nine the largest number that were made on these chickens. This covers a time range of from eight to thirteen months. Of the ten remaining birds of this group, either one suspicious or doubtful reaction or one negative reaction is recorded for one of the test periods during the experiment. In addition to these cases there were three hens in the series which were reactors consistently and showed ovarian lesions characteristic of pullorum disease but from which no organisms were obtained. There was one hen in the series (342) in which an incomplete agglutination occurred in the 1:50 dilution and negative in the 1:100 dilution at the time of the first and fourth tests, and no agglutination in any of the tubes at the sixth test, but complete agglutination in all dilutions (1:25, 1:50, 1:100) at the second, third and fifth test periods. When this bird was examined after death, it showed definite evidence of pullorum disease pathologically and bacteriologically. The results show that there were six birds in which the agglutination reaction was positive a majority of the times, but in which the pathological and bacteriological findings were negative. One of these birds gave eight positive agglutinations and one negative, another seven positive and one negative, another six positive, another five, another four positive, one suspicious and one negative, and the sixth bird gave four positive, two suspicious and one negative agglutination reactions.

Throughout the entire period of the experiment, three male birds (34, P, Y) were kept in the pen with the females. These birds were tested regularly and destroyed at the termination of the experiment. At no time did any of them show a positive agglutination reaction and there was no evidence of disease observed when they were examined at necropsy.

Twenty-four hens, all giving negative agglutination records, were placed with the positive birds. These served as the "exposed" or "contact" birds. The agglutination test at each of the test periods throughout the entire experiment always remained negative in the case of thirteen of these hens. Eleven of the thirteen did not show any gross changes nor were any pathogenic organisms isolated from them when examined after death. On the other hand, two showed gross lesions of the ovary such as would be considered characteristic of pullorum infection, but *S. pullorum* was not isolated from them. The cultures from hen 537 were sterile and *Escherichia coli* was obtained from the ovary of hen 839.

A positive agglutination reaction occurred at one or more test periods in the remaining eleven hens. Hen 584 was the first to react. The first positive test recorded for this hen occurred the third month. This hen was positive also when tested in the fifth month. No test is recorded for this bird the fourth month. The hen died soon after the fifth month and, when examined postmortem, pure cultures of *S. pullorum* were isolated from the ovary. Hen 838 was another to show a positive agglutination reaction in the early months of the experiment. The first positive reaction occurred in the fourth month and it was positive when tested again the ninth and tenth months. No tests are recorded for the fifth to eighth months inclusive. This bird died in the tenth month of the experiment and, when examined postmortem, definite gross and bacteriological evidence of pullorum disease was found.

The first positive agglutination reaction recorded in the case of six of the hens (268, 362, 483, 591, 835, 1184) was nine and ten months from the beginning of the experiment. This includes the four-month period when no tests were made, and because of this lapse of testing, we have no way of knowing whether any of them would have reacted during the no-test period. The reactions continued to be positive at each succeeding monthly test of hens 483, 591, 835 and 1184. *S. pullorum* was isolated from all but 591. In the case of hen 362, a positive agglutination

occurred the ninth month, but was negative the tenth month and again positive the eleventh month. The test made in the twelfth month was negative. The gross and cultural examinations of this bird were negative. Hen 268 showed a positive reaction the ninth month, negative the tenth month, and positive again the eleventh month, and when it was examined at necropsy, characteristic ovarian lesions were found. *S. pullorum* was not isolated. The first time agglutination occurred in the case of hens F209, 251 and 302, was during the tenth month of their contact with infected birds. One of them (251) showed positive agglutination again the eleventh month, but was negative the twelfth month. No evidence of pullorum disease was found in any of them after they were destroyed.

Experiment II

Experiment II did not differ greatly from experiment I. The difference was chiefly in the fact that a more complete "pre-contact" history was known of each bird. The pre-contact history consisted of a knowledge of the reaction to the agglutination test on each bird for a period of approximately twelve months before being placed together in the same pen. The birds came into our possession in November and December of 1927, and many of them were from the same flocks as the birds used in experiment I. Throughout the pre-contact period they were in separate lots, the division being made on the basis of the agglutination reaction. Lot A contained 17 hens which were positive to the agglutination test, and two negative males. In lot B there were 17 hens and one rooster, all of which were negative to the test. The reactions were consistently positive for the hens in lot A, and consistently negative for those in lot B during the 12-month pre-contact period. In December, 1928, the two lots were placed together, which marks the beginning of the "contact" period.

In the case of the hens originally comprising lot A, the agglutination reactions remained markedly constant for the entire period of the experiment, twelve months pre-contact and nine months contact (table II). Of the seventeen birds in this lot, definite evidence of pullorum disease was established pathologically and bacteriologically in 15 (88.2 per cent). From hen 236, one of the two that did not show gross changes characteristic of pullorum infection and from which the pullorum organism was not isolated, *S. sanguinarium* was obtained. *S. aertrycke* was

TABLE II—*Results of the serologic diagnoses, bacteriological examinations for in the birds of*

BIRD	SEX	RESULTS OF SEROLOGIC											
		NOV.	DEC.	JAN.	FEB.	MAR.	MAY	JUL.	AUG.	SEP.	OCT.	NOV.	DEC.
4	F							+	+	+	+		+
48	F	+	+	+	+	+	+	+	+	+	+		+
228	F		+	+	+	+	+	+	+	+	+	+	+
230	F		+	+	+	+	+	+	+	+	+	+	+
236	F		+	+	+	+	+	+	+	+	+	+	+
245	F	+	+	+	+	+	+	+	+	+	+	+	+
255	F		+	+	+	+	+	+	+	+	+	+	+
258	F		+	+	+	+	+	+	+	+	+	+	+
277	F		+	+	+	+	+	+	+	+	+	+	+
284	F		+	+	+	+	+	+	+	+	+	+	+
294	F	+	+	+	+	+	+	+	+	+	+	+	+
312	F		+	+	+	+	+	+	+	+	+	+	+
336	F	+	+	+	+	+	+	+	+	+	+	+	+
380	M												
394	M		—	—	—		—	—	+	+	s	—	—
535	F		+	+	+	+			+	+	+	+	+
573	F		+	+	+	+		+	+	+		+	+
754	F							+	+	+	+	+	+
H932	F							+	+	+	+	+	+
1193	M							—	—	—	—	—	—
1001	F	—		—	—	—	—	—	—	—	—	—	—
1002	F		—	—	—	—	—	—	—	—	+	—	—
1003	F			—	—	—	—	—		—	—	—	—
1004	F	—		—	—	—	—	—	—	—	—	—	—
1005	F	—					—	—	—	—	—	—	—
1006	F	—					—	—	—	—	—	—	—
1008	F	—		—	—	—	—	—	—	—	—	—	—
1009	F	—		—	—	—	—	—	—	—	—	—	—
1011	F	—		—	—	—	—	—	—	—	—	—	—
1013	F	—		—	—	—	—	—	—	—	—	s	—
1016	F	—		—	—	—	—	—	—	—	—	—	—
1017	F	—		—	—	—	—	—	—	—	—	—	—
1018	F	—		—	—	—	—	—	—	—	—	—	—
1026	F	—		—	—	—	—	—	—	—	—	—	—
1031	F	—		—	—	—	—	—	—	—	—	—	—
1032	F	—					—	—	—	—	—	—	—
1033	F	—		—	—	s	—	—	—	—	—	—	—
M	M		—	—	—	—	—	—	—	—	—	—	—

F = female, M = male.
— = no agglutination.
s = agglutination at 1:25 and no agglutination at 1:50 and 1:100.
+ = agglutination at 1:25 and 1:50 and either agglutination or no agglutination at 1:100.
+ in bacteriological column = *S. pullorum* isolated.
— in bacteriological column = *S. pullorum* not isolated.
+ in pathological column = gross lesions characteristic of pullorum disease found at autopsy.
— in pathological column = gross lesions characteristic of pullorum disease not found at autopsy.

isolated from the other (hen 535). The test record of bird 394, a male bird in this lot, is of interest. This bird, after being in contact with the infected hens for nine months, gave a positive agglutination test. The reaction was again positive at the time of the tenth monthly test and, when tested in the eleventh month,

S. pullorum and observations for gross lesions characteristic of pullorum disease experiment II

Examinations								Results of Bacteriological Examinations	Results of Gross Pathological Examinations
Jan.	Feb.	Mar.	Apr.	May	Jun.	Jul.	Aug.		
+	+	+	+	+	+	+	+	+	+
+	+	+	+	+	+			+	+
+	+	+	+	+	+	+	+	+	+
+	+	+	+	+	+			+	
+	+	+	+					—	—
+	+	+	+					+	
+	+	+	+	+	+	+	+	+	+
+	+	+	+	+	+	+	+	+	
+	+	+	+	+	+	+		+	+
								+	+
+	+	+	+	+	—	+	+	+	
+	+	+	+	+	+	+	+	+	+
								+	+
—	—	—	—	—	—	—	s	—	—
—	—	—	—	—	—	—	—	—	—
+	+	+						—	—
+	+	+	+	+	+	+	+	+	+
+	+	+	+	+	+	+	+	+	+
+	+	+	+	+	+	+	+	+	
—	—	—	—	—	—	—	—	—	—
—	—	—	—	—	—	—	—	—	—
—	—	—	—	—	—	—	—	—	—
—	—	—	—	—	—	—	—	—	—
—	—	—	—	—	—	—	—	—	—
—	—	—	—	—	—	+	+	—	+
—	—	—	—	—	—	—	+	+	+
—	—	—	—	—	—	+	+	+	+
—	—	—	—	—				—	+
—	—	—	—	—	—	—	—	—	—
—	—	—	—	—	—	—	—	—	—
—	—	—	—	—	—	—	—	—	—
—	—	—						—	+
—	—	—	—	+	+	+	+	+	+
—	—	—	—	—	—	—	—	—	—
—	—	—	—	—	—	+	—	—	—
—	—	—	—	—	—	+	+	+	+
—	—	—	—	—	+	+	+	+	+
—	—	—	—	—	—	—	—	—	—

complete agglutination occurred in only the 1:25 dilution. The tests made in the twelfth month, and in each succeeding month until the termination of the experiment, were negative. There was no macroscopic pathologic evidence of infection when the bird was destroyed, and all cultures remained sterile. The

second rooster (bird 1193) was with these hens fourteen months and never showed any evidence of infection serologically, pathologically, or bacteriologically.

The birds of lot B represent the susceptibles. In this group there were seventeen hens and one rooster. All but three of the hens (1002, 1013, 1033) remained negative to the agglutination test throughout the twelve-month pre-contact period. Bird 1002 gave positive agglutinations the tenth and eleventh months of the pre-contact period. The tests were checked on a second sample of blood with the same result. Ten regular monthly tests were made on the blood of bird 1002 subsequently, one during the pre-contact and nine during the contact period. No agglutination was evident at any of these test periods. The postmortem findings also were negative. In the case of hens 1013 and 1033, the results of the serologic test warranted a "suspicious" diagnosis at one of the regular test periods. Complete agglutination occurred in the 1:25 but not in the 1:50 and 1:100 dilutions. The many preceding and succeeding negative tests (table II) recorded for each of these birds suggest the relative unimportance of this reaction. Duplicate tests were made in each case.

Six months from the time the negative hens were placed in the pen with the positive ones (lot A), hen 1018 gave a positive reaction. She was the first to show a positive agglutination. At each of the succeeding regular tests, this hen always showed complete agglutination in all dilutions. She was destroyed and examined, and the postmortem and bacteriological findings proved her to be infected with *S pullorum*. The second to show a positive reaction was hen 1033. The reaction in this case occurred seven months from the time she was in direct contact with infected birds. The reaction continued to be positive for the remainder of the experiment, and when the bird was destroyed, positive evidence of the disease was proved culturally.

Hens 1005, 1008, 1031 and 1032 showed their first positive agglutination the eighth month of the contact period, and all but hen 1031 reacted again the ninth month. This hen was negative the ninth month, and we found no evidence of disease when it was examined after death. The autopsy on hen 1005 revealed gross lesions of the ovary which are characteristic of pullorum disease, but *S. pullorum* could not be recovered in culture. The two remaining birds of this group (1003 and 1032) were definitely infected with this organism. The last of the original negative hens to show a positive agglutination was hen

1006. This hen did not react until the ninth month from the beginning of the contact period, and it was destroyed soon after. The postmortem examination revealed gross lesions in the ovary and pure cultures of *S. pullorum* were isolated from several of the diseased ova.

Ten hens and one rooster never showed any indication of infection serologically, and when they were examined bacteriologically, the causative organism of pullorum disease was not isolated.

Discussion

Is pullorum disease transmitted or does pullorum disease spread among sexually mature fowls? The answer to this question has been given in both the affirmative and negative. While most of the evidence indicates that the disease *does* spread among adult birds, when infected and non-infected fowls are kept in contact, it was proposed to obtain additional data on this question. The importance of this question is vital in the control of pullorum disease. For example, if pullorum disease is not transmitted or spread to healthy fowls when kept in an enclosure with infected ones, the necessity for the removal of the diseased is negligible. Under such conditions, all that would be necessary in the control of the disease would be to trap-nest and use eggs from only the healthy hens for hatching purposes. This makes it necessary to determine, by adequate biologic tests, which are the pullorum-infected hens. These would be identified by suitable banding, so that when they were found upon the nest, their eggs would not be used for propagation purposes. However, this situation does not exist. Pullorum disease does spread or is transmitted or disseminated among infected and non-infected adult fowls that are kept together in the same enclosures. The experiments we conducted and here reported demonstrate this conclusively.

The successful prosecution of an investigation of this kind depends upon the availability of infected and non-infected birds. The basis for determining this status of the birds was the agglutination test (tube test) for pullorum disease. Because we were particularly interested in learning whether or not non-infected fowls would contract the disease by association with infected ones, the birds which were to represent the non-infected group were of great importance. Thus the pre-contact history of the non-infected birds used in experiment II is of greater value than for those of experiment I. It will be remembered that a pre-

contact period of twelve months during which time regular monthly agglutination tests were conducted on each bird, was obtained on those representing the non-infected group of experiment II (table II). With the exception of two positive agglutination reactions occurring the tenth and eleventh months from the beginning of the experiment, in the case of hen 1002, and the reaction which suggested a suspicious diagnosis recorded at one of the test periods for hens 1013 and 1033, all birds were consistently negative throughout the entire pre-contact period. Assuming that the reaction in these cases represented an infection with the subsequent development of agglutinins, and since the agglutinins did not remain in the blood, it indicates that the infection was only transitory or temporary.

The source of infection in these cases is not known. Complete agglutination at 1:100 occurred at both the tenth and eleventh monthly tests in the case of hen 1002, but when tested the following month and at each subsequent monthly test until the end of the experiment, no evidence of agglutination was observed. In the cases of hens 1013 and 1033, agglutination occurred only in the 1:25 dilution and then only at one of the test periods. These, therefore, are of little or no significance. The case of rooster 394 is quite similar to that of hen 1002. Five agglutination tests during a period of eight months showed this bird to be negative, but at the tests made the ninth and tenth months, complete agglutination in all dilutions was observed. The test conducted in the eleventh month revealed agglutination in only the 1:25 dilution, and at the next and all subsequent test periods no agglutination occurred. The opportunities for this bird to become infected were great, because he was at all times cohabiting with infected hens.

Table II shows that the agglutination reaction was always positive at each test period on every hen in the infected group. This applies to both the pre-contact and contact periods. More than this, *S. pullorum* was isolated from 88.2 per cent of the hens representing the infected group, when they were destroyed at the close of the experiment. This gives positive assurance that pullorum infection existed in the group.

Whereas the pre-contact history on the birds used in experiment I was not nearly so complete as those in experiment II, nevertheless the subsequent tests showed fairly well that the birds selected represented non-infected and infected individuals. With the exceptions of hens 584 and 838, all selected as non-

infected did not show any evidence of agglutination for at least four successive monthly tests. The birds selected as the infected ones were, in general, consistently positive to the agglutination test throughout the experiment.

The statistical results of experiments I and II are in rather close agreement. Eleven of the 24 negative hens in experiment I and eight of the 17 negative hens in experiment II (45.8 and 47.0 per cent respectively) developed positive agglutination reactions subsequent to the time they were placed in contact with infected birds. Of the eleven hens that changed from negative to positive reactors in experiment I, six (54.5 per cent) showed positive bacteriological evidence of pullorum disease at postmortem. In experiment II, *S. pullorum* was isolated from five (62.5 per cent) of the hens in which positive agglutination occurred in the contact period. The results we have obtained show that approximately 24.0 per cent of the non-reacting and presumably non-infected birds became infected and developed the disease subsequent to association with infected fowls. This figure represents only those birds from which *S. pullorum* was isolated. If, on the other hand, we would include all those that showed positive agglutination, the percentage would be 46.3.

Conclusion

The results of this experiment show that pullorum disease *does* spread among sexually mature fowls.

References

[1]Rettger, Leo F., and Stoneburn, F. H.: Bacillary white diarrhea of young chickens. Storrs Agr. Exp. Sta. Bul. 60 (1909).
[2]Rettger, Leo F., Kirkpatrick, W. F., and Stoneburn, F. H.: Bacillary white diarrhea of young chickens. Storrs Agr. Exp. Sta. Bul. 74 (1912).
[3]Rettger, Leo F., Kirkpatrick, W. F., and Car, L. E.: Bacillary white diarrhea of young chickens. Storrs Agr. Exp. Sta. Bul. 101 (1919).
[4]Brunett, E. L.: Transmission of *Bacterium pullorum* infection among mature chickens. Corn. Vet., xviii (1928). pp. 135-150.
[5]Luttschwager, D.: Bacillary white diarrhea and its relation to fowl typhoid. Arch. f. Geflugelkrank., iii (1929), pp. 226-275.
[6]Edwards, P. R., and Hull, F. E.: The transmission of bacillary white diarrhea among hens. Jour. A. V. M. A., lxv (1929), n. s. 28 (3), pp. 333-336.
[7]Doyle, T. M.: Bacillary white diarrhea of chicks. Jour. Comp. Path. & Therap., xxxviii (1925), pp. 377-278.

Elected Honorary Associates

At a special meeting of the Council of the Royal College of Veterinary Surgeons, held during the recent International Veterinary Congress, in London, diplomas as Honorary Associates of the Royal College of Veterinary Surgeons were conferred upon Dr. George Hilton, Ottawa, Canada; Dr. John R. Mohler, Washington, D. C., and Dr. Veranus A. Moore, Ithaca, N. Y.

THE SO-CALLED LYMPHOID HYPERPLASIAS OF ANIMALS*

By WILLIAM H. FELDMAN, *Rochester, Minnesota*

Division of Experimental Surgery and Pathology, The Mayo Foundation

There are probably no diseases which lead to such confusion as those arising from the lymphoid elements or the cells of the lymphocytic series. One is bewildered by some of the discussions dealing with subjects such as lymphadenoma, pseudoleukemia, lymphoma, leukemia, lymphocytoma, alveolar sarcoma, lymphosarcomatosis, round-cell sarcoma, lymphatic leukemia, lymphosarcoma and Hodgkin's disease. To obtain a sound concept of the pathologic changes in the conditions indicated by this array of names is impossible. The writers of the past, in describing cases belonging to this group of diseases, have, with few exceptions, selected from the existing terms those which they thought most applicable to their particular cases without making any real attempt to justify the use of the terms on the basis of the fundamental pathologic changes. The literature dealing with this group of diseases as they affect animals soon reveals the fact that in many cases the diagnosis was made on the basis of a general examination only, or, if necropsy was performed, histologic study was often omitted. Furthermore, there has been relatively little opportunity for comprehensive studies of the disease in the living animal. This is not due to these diseases being of infrequent occurrence, but rather to physical reasons which have made it difficult, particularly in the larger mammals, to conduct studies for sufficient periods of time. Much valuable information has been obtained by the observations on so-called chicken leukemia, but, even here, many of the conclusions cannot be accepted entire. A great many of the observations of the past have confused rather than clarified an already badly ambiguous situation, and the opportunities for future investigation with these conditions are impressive.

In this paper an attempt will be made to describe these conditions in a unified manner in keeping with the extent of present understanding of the anatomic, pathologic and clinical characteristics.

*Read before the Twin Cities Veterinary Medical Association, St. Paul, Minnesota, March 27, 1930.

As a basis for the subsequent thesis it will be necessary to accept the hypothesis that the primary types of progressive hyperplasia of the lymphoid tissues are true neoplasms, and that they are so closely related genetically as to justify their classification in one group. This concept was first advanced by Warthin, in 1904, and has been continuously championed by him. The unrestricted growth, the invasion and destruction of normal tissues, and the widespread metastasis exhibited in the majority of these conditions are definite characteristics of a neoplastic process. This view is gaining increasing support from pathologists engaged in study of human and of comparative material, and its full acceptance would do a great deal to render intelligible these conditions.

I would group the conditions according to Mallory's classification as lymphoblastomas. Clinically and histologically, subdivisions are perhaps permissible, although, if they are used, it should be in strict conformity with acceptable established criteria. The disease often differs in its clinical manifestations. The difference is due to the anatomic situation of the primary disturbance, the degree of differentiation of the type cells, and the extent of the metastatic involvement. In other words, the different clinical varieties which may be encountered are simply different expressions of a fundamental cellular disturbance which is common to all.

Histogenesis

In a consideration of the origin of the type cell of this complex group of tumors, it is pertinent to consider the histogenesis of normal, fully mature lymphocytes. Although these cells, in their adult form, may be derived by mitosis from the lymphoid deposits throughout the body, their forerunners, according to Drinker, must be the cells arising from the hemoblast or primitive blood cell. Through developmental stages, the cells pass from a relatively immature, undifferentiated form to the fully developed large and small lymphocyte. Since the adult forms which represent the end-stage are concerned chiefly in normal physiologic processes, they obviously are not implicated in lawless proliferations which characterize the neoplastic phenomenon. Neoplasia must depend on the continuous overproduction of immature cells for which there can be no physiologic utilization, and it is these undifferentiated forms which constitute or are responsible for the type cells of lymphoblastoma.

Little, if anything, is known as to factors which may have influence on the inception of these tumors. In man it is thought that certain of them possibly may have a relation to tuberculosis[3] although definite proof of the relationship being anything more than coincidental is difficult to assemble. As far as I am aware, no significant etiologic influence has been attributed to tuberculosis in the lymphoblastomas as they affect animals, nor has any other specific infectious agent been definitely implicated in their inception. In the present state of knowledge, there seems no valid reason to suppose that the disease is due to bacteria, and about all that can safely be said of its etiology is that it is probably due to an abnormal response of certain of the lymphocytic series of cells to a stimulus of unknown origin and nature. Perhaps the excitant factor which is responsible for the progressive overproduction of the cells in this disease is the same or closely related to that responsible for carcinoma and other lawless new growths.

Clinical Varieties

Although the conditions designated as lymphoblastoma have a close genetic relationship, they exhibit some variations in their clinical and morphologic manifestations which perhaps justify at least two subdivisions.

Simple lymphoma: This is a slowly progressive, benign tumor which arises within lymph-nodes and other lymphoid tissues. It is a strictly localized growth which replaces the normal lymphatic tissue by gradual expansion. Lymphomas do not exhibit tendencies to metastasize or to infiltrate the surrounding tissues, and the leukocytic content of the blood is not disturbed. They frequently are the site of extensive retrograde changes. If such a tumor should suddenly assume malign tendencies, it no longer could be considered as a lymphoma, but would become a lymphocytoma, which is the term used to designate the malignant forms of lymphoblastoma.

Lymphocytoma: In this group should be placed all of the malignant types of neoplastic hyperplasia of which the immature lymphocyte or its forerunner is the type cell. Malignancy is evidenced by rapidity of growth, recurrence after surgical removal, infiltration and destruction of the normal tissues, multiplicity of lesions, and metastasis. Although there may be wide variations in the extent of the involvement, the main factor is the lawless and continuous overgrowth of lymphoid cells occurring

primarily in preëxistent lymphoid tissue. The cells may invade the blood stream and multiply there in such proportions as markedly to change the proportions of its cellular content. The resultant leukemic state is essentially lymphatic in nature, and the condition is often referred to as lymphatic leukemia. However, it should be emphasized that a diagnosis of lymphocytoma is not dependent on the presence of an excessive number of lymphoid cells in the blood stream, but on the neoplastic proclivities of the lymphocyte-producing tissues. In fact, perhaps in 50 per cent of the cases the customary blood picture of leukemia is lacking.

According to this view, the blood picture is of secondary importance; it is dependent on the primarily neoplastic process present extravascularly in the lymphoid tissues. The use of the words "leukemic" or "aleukemic," in a qualifying sense before the term lymphocytoma, provides a fairly concise nomenclature which is descriptive of the pathologic changes concerned.

Conditions which are now known in veterinary literature as leukemia (including the so-called leukemia of chickens), pseudoleukemia, malignant lymphadenoma, lymphosarcoma, Hodgkin's disease, and many others, should, in my opinion, all be placed under the designation of lymphocytoma.

A word here pertaining to Hodgkin's disease would not be out of place. This condition occasionally has been reported as occurring in the lower animals, although perhaps not so frequently as formerly. M'Fadyean described four cases in the dog and one in a hog. Hodgson reported one case in a hog, of which he wrote that, because the case was advanced, the diagnosis was "rendered easy." From the published descriptions of these cases, it is evident that they all had much in common with, or were identical with, the usual picture of lymphocytoma. In fairness, however, it should be mentioned that M'Fadyean was somewhat uncertain that his cases could properly be designated Hodgkin's disease, but he also wrote:

> If the condition of which these cases are illustrative cannot be enrolled under the head of Hodgkin's disease, it is equally certain that they are not covered by any other recognized term and that a new name is required for them.

Although Hodgkin's disease no doubt exists as a separate disease of the human being, and is considered by many as a granuloma with perhaps some obscure relationship to tuberculosis, and by others as a true tumor, I seriously doubt its existence in lower animals. I have never encountered a case in a

study of approximately 500 neoplasms from animals. Moreover, the descriptions of the cases recorded in the literature fail to impress one that the diagnosis was in keeping with the usual conception of what constitutes the histologic picture of Hodgkin's disease. My experience has been similar to that of Fox, pathologist to the Zoological Society of Philadelphia, who failed to see any case resembling that of Hodgkin's disease of man in the thousands of captive wild animals under his observation.

Incidence of Occurrence and Species Affected

If all species are considered, the lymphoblastomas occur with greater frequency than any of the other neoplasms affecting lower animals. In one series of 316 tumors, representing only a portion of the material in my collection, the disease was found seventy times in seven species, although in a series of 3,032 necropsies, including mammals and fowls, Crocker listed only two cases that could be classed with lymphoblastoma and both of these occurred in dogs. Crocker's figures were compiled from records of a veterinary hospital, whereas much of my material was from slaughter-houses.

Practically all domestic species of animals are susceptible to the disease, although unquestionably it occurs more frequently in some than in others. As early as 1858, Leisering, who first recognized leukemia in animals, stated that it is more frequent in dogs than other house animals, and certainly the literature on the subject contains more references to this animal than to any other. Here, as is the case with all tumors of animals, it is extremely difficult to obtain accurate figures on the occurrence of the disease in the respective species, because of difficulty in collecting the material, as well as to the paucity of published data that are reliable.

The before-mentioned seventy cases in my series were distributed among the various species as follows: bovine, twenty-three; dog, eleven; horse, one; fowl, seventeen; sheep, four; swine, thirteen, and rabbit, one.

Crocker's two cases in dogs occurred in a total of 1,548 dogs examined, which certainly does not represent a high rate of incidence. The disease has been observed in cats by Siedamgrotsky and by Lellmann. Houdemer and Bablet described the only case I have found of generalized lymphoblastomatous involvement of the abdominal cavity and viscera in a mule.

It is of interest to note that the disease occurs not infrequently in mice, and Simonds, in studying material from the first 15,000 necropsies of Slye's stock, found primary malignant lymphoid hyperplasia in 190 of the animals.

The material I have received during the last two years, much of which does not appear in the statistics given, indicates that the disease is extremely common in the bovine species. It is familiar to all veterinarians in the federal meat inspection service of the Bureau of Animal Industry, where it is diagnosed routinely as leukemia. The reports of the chief of the Bureau of Animal Industry of the United States Department of Agriculture for three years prior to August 24, 1929, show "leukemia" to have been the cause of condemnations among the various classes of live stock as given in table I.

TABLE I—*Condemnations for leukemia by federal meat inspection service of the Bureau of Animal Industry**

ANIMAL	NECROPSIES	ANIMALS CONDEMNED FOR SO-CALLED LEUKEMIA	RATIO
Cattle	27,373,941	2918	1 in approximately 9,000
Calves†	14,380,473	83	1 in approximately 173,000
Sheep	39,646,722	18	1 in approximately 2,000,000
Swine	158,161,409	650	1 in approximately 212,000
Horses	266,277	2	1 in approximately 133,000

*During the period over which these statistics were gathered necropsies were also performed on 72,117 goat carcasses in none of which was there gross demonstrable evidence of "leukemia."
†Bovine animals one year or less in age.

From the federal meat inspection reports, it is impossible to determine the total number of true neoplasms encountered in slaughtered animals, since the diagnosis usually is made at necropsy from the gross aspects of the condition. The routine is such that unlimited time is not available to make a thorough study of each and every formation resembling a tumor. As a consequence, many abnormalities other than neoplasms are grouped with the true tumors. For this reason it is difficult to compare the incidence of the various kinds of neoplasms. Likewise is it impossible to separate most of the cases of so-called leukemia in the literature from those in which the disorder was of a myeloid nature, with which this paper is not particularly concerned. Myeloid conditions seem to occur infrequently in the lower animals, and although histologic study would be necessary in order definitely to identify many of the cases, the

myeloid variety generally does not exhibit the predilection for the lymphoid tissues so characteristic of the lymphoblastomas.

The facts seem to justify the conclusion that primary lymphoid hyperplasia of a neoplastic nature is among the more common diseases of many of the domestic animals and undoubtedly is the cause of many more or less obscure deaths, for unless a thorough necropsy is performed, the true nature of the disease may escape detection. From the available data, it seems that the disease most commonly affects dogs, cattle and chickens.

Symptoms

It is difficult to describe what might be considered typical symptoms of lymphoblastoma as it occurs in animals. This is especially true of the early stages, because of the insidious onset of the disease. In many of the cases reported, there has been noted a tendency to tire easily, associated with an increased appetite, although in cases in which the digestive apparatus is involved there is usually noticeable anorexia, bloating, and often diarrhea. There is frequently progressive loss of flesh, and if the disease continues sufficiently long before the death of the animal, a state of emaciation may result. The animal is usually disinclined to move, and appears listless and disinterested in its surroundings. The appearance of the disease in cattle which are lactating very often occasions sudden cessation of the flow of milk. As the disease progresses, and the animal becomes weaker, it prefers a recumbent position. If the heart is involved, as so often is the case, cardiac irregularities may be detected; frequently the animal has difficulty in swallowing and symptoms of respiratory distress are not uncommon.

The palpable lymph-nodes are often enlarged and firm, but are not painful. The involvement of lymph-nodes is frequently bilateral and those of the head and cervical region are usually the first to become noticeable, especially in dogs and cattle. However, I have observed cases in which lesions of the disease were extensively present in the thoracic and abdominal viscera, but without any apparent increase in the palpable lymph-nodes of the body. Such cases are exceptional.

The course of the disease is variable. An occasional case may terminate fatally within a week or so after the recognition of symptoms. In the majority of cases, the disease is of longer duration, and in some cases it may continue for many weeks, or even for a few months, before death ensues.

Pathologic Anatomy

Since this is a disease primarily of the lymphoid tissue, one naturally expects lesions to be present in these structures. When one realizes the extent of the distribution of lymphoid tissue through the body, the widespread distribution of the lesions frequently seen in this condition is not remarkable. Although the lesions may be confined to the lymph-nodes and the other deposits of lymphoid tissue, the disease is capable of extensive metastasis, and secondary involvement may occur in the lungs, heart (fig. 1), liver, kidney, and even in the brain. Peculiar as it may seem, and contrary to the opinion of many, the spleen, in many of my cases, was not demonstrably involved. Although

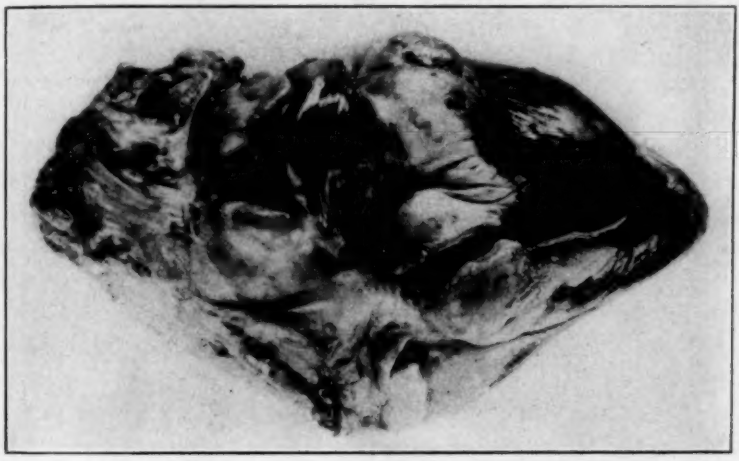

Fig. 1. Heart of a nine-year-old cow. Numerous nodules involve the cardiac musculature. Large masses of tumor tissue were present also in the omentum, along the intestines, and in the uterus. The clinical diagnosis was "cardiac disturbance"; the microscopic diagnosis was aleukemic lymphocytoma.

the cardiac muscle and the voluntary striated muscles often are extensively invaded metastatically, smooth muscle seems to possess a more or less stubborn resistance to the advances of the neoplastic process (fig. 2). A summary of the lesions which occurred in a series of twenty-one unselected cases in bovine animals, nineteen of which were female, showed the following: The spleen was affected in seven; the liver in five; the lungs in eight; the digestive tract in nine; the uterus in eight; the cardiac muscle in twelve, and the blood was leukemic in seven, as determined by cross sections of blood-vessels. In none of these was the parenchyma of the kidney violated, although in several animals extensive neoplastic masses were present in the sub-

capsular region of this organ. There was extensive involvement of lymph-nodes in all but two of the cases, and in one case no gross lymphatic abnormality was discernible. Unfortunately, in this case no material was available for histologic study of the lymph-nodes; the diagnosis was made on the lesions as they affected the

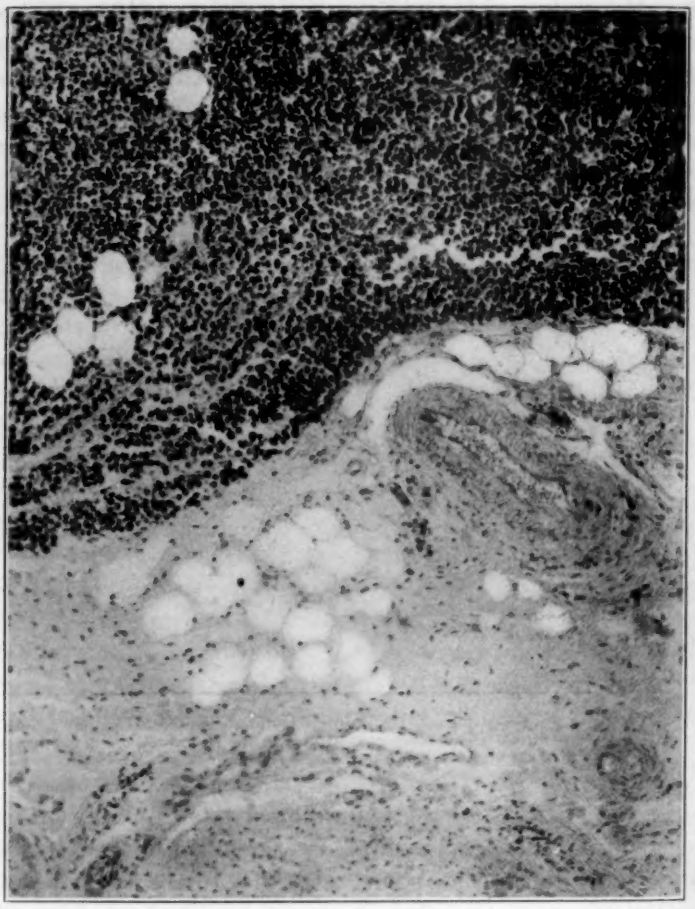

FIG. 2. Abomasum of a nine-year-old cow. The sharp line of demarcation between the tumor cells and the muscular layer is shown. A diagnosis was made of leukemic lymphocytoma (x200). (See also fig. 6.).

other tissue, particularly the heart. Many of the affected lymph-nodes in this group of cases were extremely large; one weighed eight pounds, and in another case the mesenteric group weighed thirty pounds. In many cases the lesions, which were relatively small and multiple, occupied the subserosa of the intestines or

the subperitoneal region of the abdominal wall or diaphragm (fig. 3). In one case in my series the eyeballs projected from their normal position in a most striking fashion, because of the presence in the orbits of large masses of tumorous tissue. Knuth and Volkmann observed similar involvement of the orbit in five of the nineteen cases in bovine animals studied. Edema of many of the tissues adjacent to the neoplastic tissue occasionally was present. Although the kidney was not involved in any of the twenty-one cases before mentioned, I did find metastatic tumors in this organ in one bovine animal. The animal was a Shorthorn

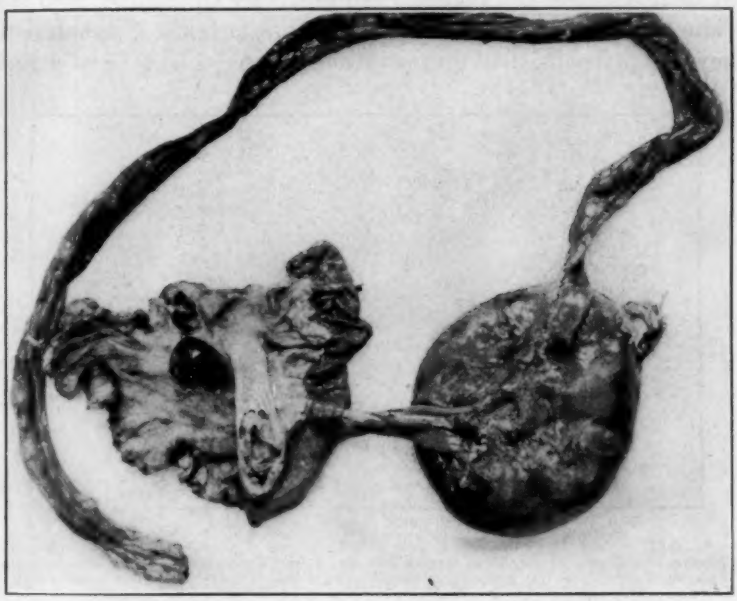

FIG. 3. A portion of the small intestine of a six-month-old hog. The large tumorous sacculation which occurred as a consequence of the neoplastic involvement of the intestine is shown. Many small nodular tumors were present along the intestine, one segment 75 cm. long possessing twenty of these smaller structures. The liver and spleen were involved subserously. A diagnosis was made of leukemic lymphocytoma.

bull, five years old, which had been treated two years before death for actinomycosis. At necropsy, there was marked enlargement of the mesenteric lymph-nodes, and several of the regional body lymph-nodes also were involved. One kidney contained a number of grayish-white, spherical nodules, measuring up to 1.5 cm. in diameter, and confined largely to the cortical portion of the organ. Many of the nodules projected slightly above the surface in a dome-like manner, but the renal capsule was intact and apparently normal.

The organs affected differ greatly in the various species. In swine the kidneys are frequently affected (fig. 4), whereas in sheep the distribution of the lesions is similar to those in the ox. In chickens the liver, spleen and kidneys are the organs of predilection. Although tumors may develop in the tissues of the head or of the neck, or in the subcutaneous tissues anywhere on the body, in the common fowl, multiple, large, irregular, neoplastic masses may also occur in the walls of the intestine. In the dog, the disease not uncommonly arises as small, nodular growths in the skin or subcutis of the neck, shoulder, or thoracic region, and metastasis to the regional lymph-nodes frequently results.

The gross appearance of these tumors is fairly characteristic. They usually consist of diffuse, fleshy masses of tissue of a flesh-

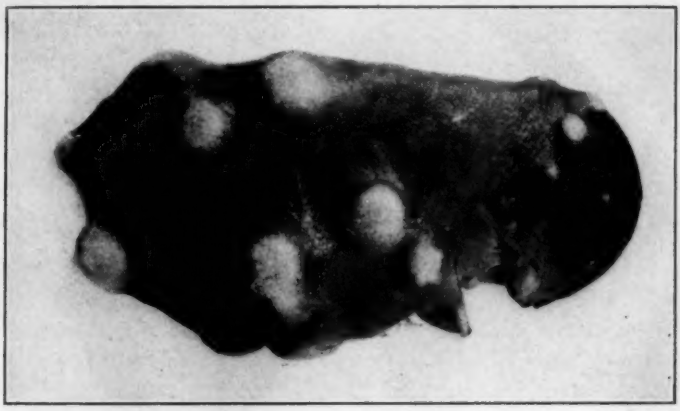

FIG. 4. Kidney of a seven-month-old hog in which were multiple tumorous nodules. Both kidneys, the liver and the lungs were affected. Tumors were present also on the costal pleura, and in the left prepectoral region there was a large tumorous mass which was undergoing necrosis. A diagnosis was made of leukemic lymphocytoma.

pink or grayish-white color. They are often somewhat putty-like in consistence. They are usually of a compact texture, but are not hard. Degeneration in various stages is frequently seen. Microscopically the lymphoblastoma presents a fairly typical picture.

Lymphoma: These tumors consist of rather sharply circumscribed collections of lymphoid cells without the features usually associated with the appearance of malignancy. The cells are of a fairly uniform size, and mitosis is seen only occasionally. There is, however, unmistakable evidence of slow progressive encroachment on the surrounding lymphoid tissue. The chronicity of these tumors is favorable for the appearance of retrogressive changes,

and in one case which I studied much of the tumor substance was involved in atypical amyloid degeneration.

Lymphocytoma: The type cell of these tumors has been designated "round cell" by many of the older writers, and either the large or small variety may predominate. The cell, which is lymphoid in nature, is irregularly spherical, with a narrow zone of non-granular cytoplasm surrounding a slightly eccentrically situated nucleus. The nucleus, which constitutes most of the cellular bulk, is spherical and is strongly basophilic. An abun-

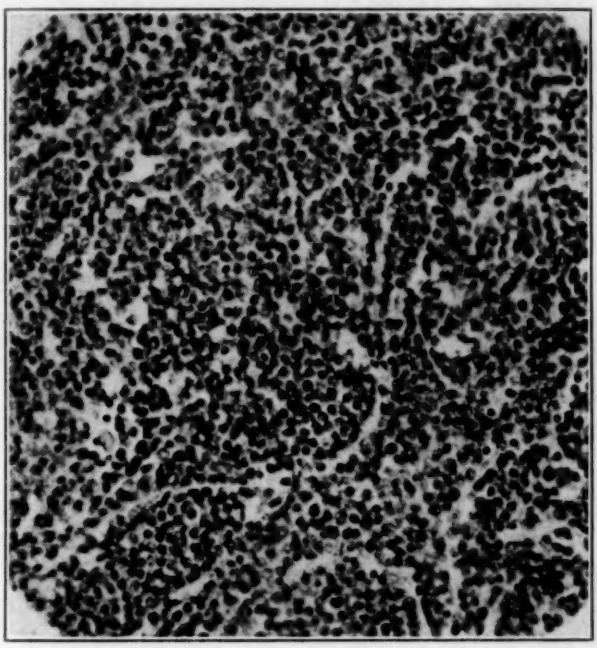

FIG. 5. Aleukemic lymphocytoma situated adjacent to the kidney of an eight-year-old cow. The heart was extensively involved, but the large lymph-nodes of the body were not grossly affected. The compact masses of lymphoid cells, many with hyperchromatic nuclei, are characteristic of these tumors (x220).

dance of chromatin is distributed as small granules throughout the nuclear substance. Nucleoli are seldom observed. Mitosis is often a striking feature of this type of neoplasm, and frequently several cells showing different phases of this phenomenon can be observed in a single field. The cells vary considerably in size, as do those of most rapidly growing malignant tumors. This is particularly characteristic of cells which are immature and undifferentiated.

The cells are laid down in compact formation, occasionally with a tendency toward an alveolar type of architecture. A few delicate strands of fibrous stroma may be demonstrable, but this material is usually scant in the more malignant forms (fig. 5). Definite blood-channels seldom are present; the blood appears to be in direct contact with the cells of the tumor. In the larger specimens small regions of necrosis occasionally may be present.

When lymph-nodes are invaded, the cells of the tumor soon replace the lymph tissue, and sections often reveal only the slightest traces of the original structure. Involvement of the musculature causes destruction of the muscle fibers, their replacement by compact masses of tumor cells, and hydropic degeneration often is seen in the muscle fibers adjacent to an area of invasion. In the lungs and spleen the tumorous process appears as diffuse masses of closely packed cells which replace the normal parenchyma, and there is no demonstrable attempt

FIG. 6. A cross-sectional view through the wall of the abomasum of a nine-year-old cow, showing the neoplastic tissue between the mucosa and the muscular coat. The wall of the organ was 3 cm. in thickness. The heart was extensively involved. A diagnosis was made of leukemic lymphocytoma. (See also fig. 2.).

by the invaded tissues to encapsulate the invading tissue. In the affected liver, definite foci of neoplastic cells are commonly seen (fig. 6), whereas in the digestive tract and uterus the majority of tumorous cells are confined to the submucosa, which may be enormously expanded if the involvement is extensive (fig. 7).

In those cases in which there is invasion of the blood stream by the cells of the tumor, sections of the liver usually will reveal variable numbers of tumorous cells in the capillaries of the hepatic sinusoids (fig. 8). Cross sections of larger vessels, if they contain blood, usually will disclose an excessively large number of lymphoid cells, which makes it possible to determine a leukemic state in many cases, even though smears of blood were not obtained during life.

Smears of blood from animals in which the blood is leukemic reveal diminution of the concentration of hemoglobin, slowing

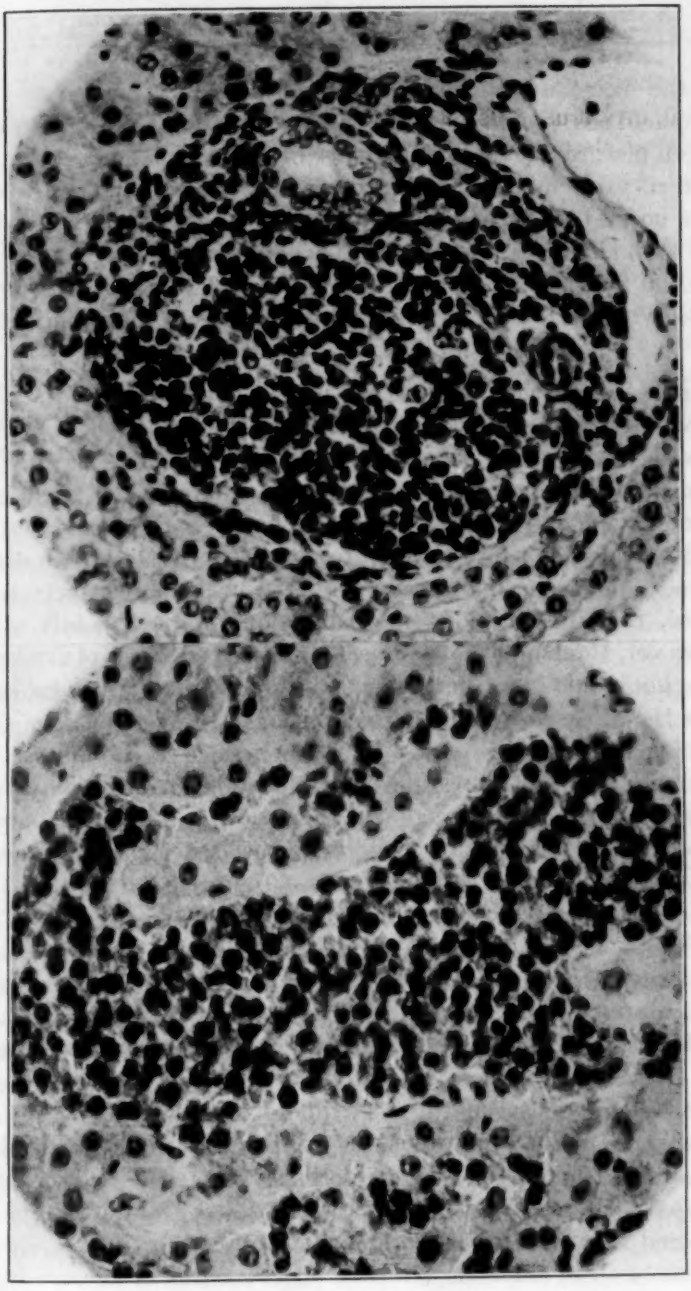

FIG. 7. (Above) Solitary nodule of neoplastic cells in the liver of a two-year-old cow. The neoplastic process involved the entire cervical and outer anterior thoracic region. The prescapular lymph-nodes were greatly enlarged. The liver was not grossly affected. A diagnosis was made of leukemic lymphocytoma (x380).

FIG. 8. (Below) The liver of a ten-year-old cow. A blood-vessel filled to capacity with neoplastic lymphoid cells is shown. The smaller sinusoids of the liver likewise possessed a large number of similar cells, an example of circulating metastasis secondary to the primary situation of the disease. The heart, uterus and small intestine also were affected. A diagnosis was made of leukemic lymphocytoma (x500).

of the coagulation time, and nucleated erythrocytes. The lymphoid cell, in various stages of its development, dominates the blood picture so that the ratios between the leukocytes and erythrocytes, instead of being approximately 1 to 400 or 1 to 600, as in normal blood, may be as low as 1 to 25 or 1 to 50. In cattle the total leukocyte count may vary between 100,000 to more than 200,000 for each cubic millimeter of blood, whereas in dogs the leukocyte count may reach 320,000 for each cubic millimeter.[16]

Influence of Age, Sex and Breed

In twenty of the bovine cases of lymphoblastoma in the series previously mentioned, the age of the animals ranged from eight months to ten years. Of one animal the age was not known. The animals were distributed among the different age periods as follows: under one year, one case; between one year and five years, five cases, and between five years and ten years, fourteen cases. As far as one can conclude from this relatively small series, the disease shows a predilection for older adult cattle. However, Udall and Olafson recently described a case of aleukemic lymphocytoma in a Guernsey calf aged six weeks, which indicates that the disease occasionally may occur in the very young. Concerning the influence of age on the incidence of the disease, the statistics of meat inspection previously referred to are of particular interest. Although the disease was observed once in approximately 9,000 of the bovine species designated as cattle (more than one year of age), it was found in calves (less than one year of age) once in approximately 173,000.

The paucity of cases among sheep and swine perhaps can be accounted for by the fact that the majority of these animals are slaughtered when young or in early adult life. The extreme rarity of the disease in horses, and its apparent absence in goats, suggests the possession by these species of marked lack of susceptibility to neoplasia of this nature. Although no particular study has been made of the relation of age to the disease as it affects dogs, my observation would lead me to believe that the relationship is comparable with that in cattle and that the majority of the affected animals are in middle or late adult life. The disease is seldom seen in chickens less than eight months of age.

Although all but one of the twenty-one bovine cases included in this report occurred in females, I do not think that sex is of significance in the frequency of the disease. It occurs most frequently in the female because more of this sex are permitted to attain adult life than males. This is true also of chickens.

It is difficult to believe that breed has any influence on the incidence of lymphoblastoma in the respective species, although the available data are entirely inadequate to permit definite conclusions to be made. In nineteen of the bovine cases before mentioned, in which the breed was known, the disease affected Shorthorns ten times and Holsteins six times, whereas Jerseys, Guernseys, and animals of the Angus breed were each affected once. The respective breeds are perhaps represented in about the same proportion as they are presented for slaughter, although the apparent rarity of the disease in the Hereford breed cannot be explained on this basis.

Diagnostic Considerations

Of chief importance in the diagnosis of lymphoblastoma is the sudden appearance of tumorous enlargements, which cannot be accounted for by infection or trauma. Obscure nodular swellings in the skin of the neck or thoracic region of dogs should be considered possible lymphoblastomas, and if they persist, biopsy should be made in order to enable a histologic diagnosis. Particular attention should be given to the more important lymph-nodes of the body, to determine whether they are enlarged, a bilateral enlargement being especially significant of the presence of lymphoblastoma. A history of increasing fatigue, preference for a recumbent position, and evidence of digestive disturbances should be looked on as suggestive of the disease. Enlargement of the spleen is of doubtful importance. If examination of the blood reveals a leukemic state in which the leukocytes are predominantly lymphoid in nature, a diagnosis of leukemic lymphocytoma is suggested, and confirmatory lesions usually can be demonstrated. It should be kept in mind, however, that demonstrable changes in the blood are absent in a large percentage of cases and may be transitory in others, and as a consequence the importance of the blood picture is diminished in the diagnosis of this condition. However, for its separation from myelogenous leukemia and so-called chloroma, a study of the blood is most important. These conditions are not so frequent in their occurrence in the lower animals as are the lymphoid disturbances. In fact, their rarity makes the recognition of a case noteworthy pathologically.

In the differentiation of lymphoblastoma and myelogenous leukemia and its closely related condition known as chloroma, the green color of the tumorous tissue in chloroma is characteristic

and should suggest the true nature of the condition. However, the final diagnosis must depend on the results of cytologic studies of the blood and tumorous tissue, the details of which cannot be considered here. There are other conditions which may be confused with lymphoblastoma during the general examination.

In dogs there are cutaneous granuloma,[4] transmissible (so-called infectious) lymphosarcoma, which occupies a special place in the classification of tumors, and tuberculosis and other metastatic tumors which may invade the lymph-nodes.

In bovine animals tuberculosis should be excluded in the differential diagnosis of lymphoblastoma, and the possibility of the coexistence of the two diseases should be considered. The tuberculin test would be helpful in determining this point, as would biopsy if it could be obtained. In some regards, individual cases of lymphoblastoma of cattle may suggest traumatic pericarditis. This is most likely to happen in those more or less acute cases in which evident enlargement of lymph-nodes does not occur. However, in traumatic pericarditis there is usually more or less fever, and significant polymorphonuclear leukocytosis, which are features foreign to neoplastic disease. The friction sounds in the cardiac region, together with the splashing of the pericardial fluid, so often characteristic of traumatic pericarditis, are likewise absent.

In sheep, enlargement of lymph-nodes due to non-specific pyogenic bacteria, as well as the enlargement of caseous lymphadenitis, may be confused with this condition. However, the disease is so seldom seen in this species as to make the necessity of a diagnosis during life improbable. Swine likewise are so rarely affected as to make the disease of negligible importance in the clinical diagnostic consideration of the diseases of this animal.

Treatment

The surgical treatment of simple lymphomas situated in accessible situations should be of permanent benefit.

The lymphocytomas, however, which are of a malignant nature, cannot be considered as amenable to surgical interference in animals. Even when the operation is done early, and most thoroughly, recurrence, with metastasis, may be expected. The widespread distribution of most cases of lymphocytoma precludes the possibility of successful surgical intervention. In human beings affected with the disease, Fowler's solution, sodium cacodylate, and, more recently, arsphenamine, have been used as

treatment for the resultant anemia. For the destruction of the abnormal lymphoid cells, Roentgen rays, benzol and radium have been used. Although temporarily promising results are frequently observed following the therapeutic application of these agents, the disease is practically always fatal.

Conclusions

Incomplete and fragmentary data indicate that so-called lymphoid hyperplasia constitutes an important group of related conditions of rather frequent occurrence in the lower animals. Although practically all domestic species are susceptible, the disease is seen most often in cattle, dogs and chickens.

Genetically and histologically there is sufficient similarity between the various manifestations of the disease to justify the conclusion that they represent different expressions of a process which is fundamentally identical. Their obscure inception, unrestrained growth, destructiveness, widespread metastasis, tendency to recur after removal, and resistance to all forms of treatment make the neoplastic concept of this disease acceptable.

A simplified classification is desirable and, following Mallory's suggestion, all forms of primary neoplastic lymphoid hyperplasia are placed under the general heading of lymphoblastoma. The benign forms are called lymphomas, and those with malignant tendencies are designated lymphocytomas, leukemic or aleukemic, depending on the presence in the blood of circulating metastasis.

The word leukemia should be used only in a qualifying sense, since the leukemic state, when it is present, is not the primary expression of the disease. It is a secondary expression, dependent entirely on the lawless activities of the lymphoid tissues of the body for its inception.

There is no proof, histologically or otherwise, that the condition known as Hodgkin's disease exists in the lower animals.

Anatomically, lymphoblastomas may arise locally or systemically, and metastasis commonly results. In the majority of cases there is noticeable enlargement of lymphoid tissues.

Breed and sex seem to be without influence on the occurrence of the disease. The effect of age is suggestive, since in the limited observations made, the majority of cases appeared in older adult animals.

Clinically, the disease may be confused with certain non-neoplastic swellings of the lymph-nodes, particularly tuberculosis.

Little can be offered of permanent benefit in the treatment of the malignant lymphoblastomas, and the disease is invariably fatal.

REFERENCES

[1]Crocker. W. J.: Three thousand autopsies. Corn. Vet., ix (1919), pp. 142-161.
[2]Drinker, C. K.: Diseases of the Blood. Oxford Medicine (Oxford University Press, New York, 1920), II, pp. 510-580.
[3]Ewing, James: Neoplastic Diseases. A Treatise on Tumors (3rd ed.; W. B. Saunders Co., Philadelphia, 1928), p. 421.
[4]Feldman, William H.: Kennel granuloma. Jour. A. V. M. A., lxxiii (1928), n. s. 26 (5), pp. 617-622.
[5]Fox, Herbert: Diseases in Captive Wild Mammals and Birds. Incidence, Description, Comparison (J. B. Lippincott Co., Philadelphia, 1923), p. 118.
[6]Hodgson, J. F.: Hodgkin's disease in a pig. Jour. Comp. Path. & Therap., xvi (1903), p. 382.
[7]Houdemer and Bablet, J.: Deux cas de tumeurs ganglionnaires malignes observées au Tonkin chez des animaux domestiques. Bul. Soc. Path. Exot., xx (1927), pp. 345-349; Abstract. Cancer Rev., ii (1927), p. 402.
[8]Knuth, P., and Volkmann: Untersuchungen uber die Lymphozytomatose des Rindes. Ztschr. f. Infekt., xvii (1915), pp. 395-467.
[9]Leisering, A.: Quoted by Ordway, Thomas and Gorham, L. W.: Leukemia. Oxford Medicine (Oxford University Press, New York, 1920), II, pp. 682-757.
[10]Lellman: Quoted by Simonds.
[11]Mallory, F. B.: The Principles of Pathologic Histology (1st ed.; W. B. Saunders Co., Philadelphia, 1914), p. 326.
[12]M'Fadyean, J.: Five cases of Hodgkin's disease in the lower animals. Jour. Comp. Path. & Therap., xvi (1903), pp. 379-382.
[13]Siedamgrotzky: Quoted by Simonds.
[14]Simonds, J. P.: Leukemia, pseudoleukemia and related conditions in the Slye stock of mice. Jour. Cancer Res., ix (1925), pp. 329-373.
[15]Udall, D. H., and Olafson, Peter: Pseudoleukemia in a calf. Corn. Vet., xx (1930), pp. 81-84.
[16]Warthin, A. S.: The neoplasm theory of leukemia, with report of a case supporting this view. Trans. Asso. Amer. Phys., xix (1904), pp. 421-432. Diseases of the Lymphatic Glands. Osler's Modern Medicine (Lea and Febiger, Philadelphia, 1927), V, pp. 199-225.
[17]Weil and Clerc: Quoted by Hutyra, Franz, and Marek, Josef: Special Pathology and Therapeutics of the Diseases of Domestic Animals (3rd ed.; Alexander Eger, Chicago, 1926), III, p. 126.

Obtain California Licenses

The following veterinarians were granted licenses to practice in California, as a result of the examinations held by the California State Board of Veterinary Medical Examiners, June 17, 1930:

Drs. J. A. Campbell (K. C. V. C. '17), Williamsfield, Ill.; J. E. Clair (K. S. A. C. '30), Burbank, Calif.; Robert Darling (Mont. '89), San Diego, Calif.; C. S. French (O. S. U. '11), Pacific Palisades, Calif.; Neil J. C. Halpin (Wash. '29), Pasadena, Calif.; F. T. Harris (Wash. '28), Twin Falls, Idaho; L. O. Lietzman (Ind. '24), Whittier, Calif.; R. J. McWherter (Wash. '29), Los Angeles, Calif.; John K. Perry (Ga. '29), Hollywood, Calif.; M. B. Royer (U. P. '11), Glendale, Calif.; W. F. Sexton (Wash. '30), Riverside, Calif.; Donald E. Stover (Wash. '30), Long Beach, Calif.; C. C. Sundstrom (Colo. '30), Santa Monica, Calif.; M. A. Thom (Wash. '29), Los Angeles, Calif.; V. Todorovic (Colo. '27), Tennant, Calif.; J. G. Townsend (Corn. '19), Los Angeles, Calif.; H. G. Tully (Corn. '29), Hollywood, Calif.; J. E. Wilson (Colo. '30), Santa Monica, Calif.

RECENT ADVANCES IN THE PHYSIOLOGY OF DIGESTION

II. The Gastric Glands

By H. H. DUKES, *Ames, Iowa*

Department of Veterinary Investigation, Iowa State College

The mucous membrane of the simple stomach presents the following gland regions: cardiac, fundus and pyloric. In some animals an additional non-glandular zone known as the esophageal region is found. In the horse, as is well known, the esophageal region is large; in the pig it is small. No glands are found in this region, though the stratified squamous cells covering it produce some mucus. The fundus glands are the gastric glands proper.[1] The pyloric glands (dog) produce a scanty, viscid, slightly alkaline secretion containing mucus and a proteolytic enzyme only one-twenty-fifth as active as pepsin secreted by the fundus glands.[2] The significance of the cardiac glands, especially in the pig, in which animal they occupy an extensive zone, has been the subject of considerable controversy. Bensley,[3] who has made an extensive study of the cardiac glands, is of the opinion that they are mucous glands and therefore do not produce enzyme. According to this worker they are to be regarded as retrogressive structures derived from the fundus glands and destined ultimately to disappear. The glandless esophageal zone in the horse, and the glandless rumen, reticulum and omasum of the ruminant are, on the basis of this view, to be regarded as regions from which gastric glands have entirely disappeared in the course of evolution, the stomach, in the case of the ruminant, at the same time having undergone extensive modifications in form. If these views are correct, one is to believe that the horse and pig are losing the glands of the stomach; the glands of the esophageal zone have already disappeared and those of the cardiac zone are disappearing. How far will the process extend into the fundus-gland region? Will the horse and pig, and perhaps all Herbivora, develop ultimately something akin to the ruminant type of stomach?

Ellenberger and Scheunert[4] report that, contrary to their earlier view, the cardiac glands of the pig do not secrete an amylase. This was determined from studies on the secretion obtained from

Received for publication, April 12, 1930.

an experimentally-produced fistulous pouch of the cardiac-gland zone.

Gastric Juice

Gastric juice in pure form has never been obtained from solipeds nor from pigs, though it has been obtained from several of the smaller simple-stomached animals, including the dog, cat and rabbit; from the calf and goat; and from fowls. In all animals studied there is a striking similarity in the composition of the juice. Pepsin and hydrochloric acid appear to be always present. Rennin may be absent from the stomach of adult mammals that do not receive milk in their diet. As far as the evidence goes, it appears that the concentration of hydrochloric acid is somewhat greater in the gastric juice of carnivores than of herbivores. Thus, in the pure juice of the goat, its concentration is 0.044 per cent[5]; of the calf, 0.13 to 0.356 per cent[6]; of the goose, about 0.27 per cent[7]; and of the carnivorous dog, about 0.55 per cent.[8] However on the basis of such figures as these, there is little justification for the popular belief that the ability of the dog to digest bones is due primarily to the strong acidity of its gastric juice. Some other factor or factors must operate.

Gastric lipase is most abundant in carnivores and rodents, almost absent in fish and birds, and practically absent in ruminants. The optimal reaction for its activity in different animals is as follows[9]:

Optimal pH for gastric lipase

Dog	6.3
Cat	5.5
Rabbit	6.3
Pig	7.1
Sheep	6.3
Horse	7.5 (av. of 7.1 and 7.9)

The Control of the Secretion of Gastric Juice

Recent work on the control of the secretion of gastric juice is extensive. The animal used in most experiments was the dog. No attempt is made here to give complete citations. Further references will be found in connection with the articles.[10, 11, 12, 23] The following is a brief summary of the work:

Gastric secretion is under the control of stimuli operating from three parts of the alimentary canal. We may speak therefore of a psychic or cephalic phase, a gastric phase, and an intestinal phase. The psychic phase has long been recognized. During its operation, stimuli reach the gastric glands, *via* the vagi, as a result of the sensations of eating, as for example when an animal

with a divided esophagus and gastric fistula is fed (sham feeding). In view of the fact, however, that a decerebrate dog will secrete gastric juice when sham-fed, it is clear that the control is not entirely cerebral; hence we may speak of a cephalic phase. The psychic phase is said to be absent in ruminants.

When food enters the stomach a more copious secretion takes place. This is the gastric phase and is due to two sets of stimuli, mechanical and humoral. Mechanical stimuli are due to the contact of food with the mucous membrane and the result may be a reflex stimulation of the gastric glands, or a local increase in their blood supply, or both. Humoral stimulation signifies the existence of chemical excitants in the blood-stream. This method of control of the gastric glands has long been believed to exist, but positive proof was not obtained until recently, when it was shown that a pouch of the stomach transplanted under the skin would secrete when the animal was fed. Clearly, when food was consumed, something entered the blood-stream and was carried to the transplanted pouch which was stimulated to secrete. Whether the humoral excitant is a hormone, *i. e.*, whether it is actually produced by the body, or whether it is preformed in the food or formed from the food during digestion, was not determined. The substance has more recently been shown[14] to be present in a dialysate obtained by passing the circulating blood of a dog through a special kind of dialyzer(vividialysis).

That an intestinal phase exists is shown when a dog, whose esophagus is anastomosed directly to the duodenum and whose stomach is connected to the outside by a fistula, secretes gastric juice, after a time, when fed. The food in this case goes directly into the intestine, yet the stomach secretes. Obviously some form of stimulation arising in the intestine must reach the stomach.

Bacteria and Plant Enzymes

In the stomach of Herbivora bacteria[15] and plant enzymes[16] begin their work. As hydrochloric acid accumulates, their action is inhibited. It is likely that both of these agencies are more important in digestion in Herbivora than is usually appreciated.

The Filling of the Stomach

In herbivores there is a marked tendency for food to stratify in the left extremity and body of the stomach. This condition has been studied for the horse[17] and pig[18] by feeding animals with a series of artificially colored foods and then killing the animals, removing the stomach, and freezing and sectioning it. In this

way the stratification could be noted. Mixing takes place only in the pyloric part of the stomach.

In view of this well-known tendency of food to stratify in the stomach of the horse, and in view of the fact that the stomach is not large enough to hold an ordinary meal and any considerable amount of water, the question arises as to the effect on gastric digestion of watering horses when the stomach is full. Observations by several investigators agree in showing that the arrangement of food in the stomach of this animal is not seriously disturbed by water-drinking and that little or no washing out of food from the stomach occurs.[17, 19, 20]

How Important is Stomach Digestion in the Horse?

The stomach of the horse is so small that an animal may swallow, during a given meal, several times the volume of material (food and saliva) that remains in the stomach at the close of the meal. The excess must pass on into the intestine during the meal. This naturally means that a good deal of food cannot remain long in the stomach. This fact has been recognized since the time of Colin. By virtue of this consideration and the view of Bensley[1] that the esophageal zone represents an area from which gastric glands have disappeared and that the cardiac glands are partially degenerated fundus glands, one naturally asks, "How important, after all, are the digestive changes that take place in the horse's stomach?" A definite answer cannot be given as yet; nevertheless one gains the impression that they cannot be of prime importance.

While the views suggested here tend to minimize the importance of the digestive changes taking place in the stomach of the horse, sight is not lost of the fact that clinically digestive disorders of stomach origin are common and important. Perhaps the explanation for these disorders is due not alone to errors of management but in part to the transition through which the stomach appears to be going.

The Control of the Pylorus

The theory of acid control of the pylorus[21] is now considered to be largely disproved. According to Alvarez,[22] anything that stimulates the stomach will cause it to empty faster, while anything that stimulates the upper part of the intestine will delay its emptying. Thus hydrochloric acid becomes only one of a number of factors concerned in the emptying of the stomach.

Stomach Movements

We have little direct information concerning stomach movements in the horse and pig. In any animal it is possible to insert a balloon and rubber tube through the esophagus and into the stomach, and thus to record the movements of the viscus on the smoked paper of a kymograph, but in large animals it is difficult or impossible to determine the location of the balloon and hence difficult or impossible to interpret the tracing.[23] In the case of small animals, however, the location of the balloon can be determined by means of the Roentgen ray. Numerous other methods of studying stomach movements are applicable to the small animals, but so far they appear not to have been extended to solipeds and the pig. Insurmountable difficulties may exist with certain methods in case of the horse, but there seems to be no good reason why some of them, for example, the open-abdomen technic of Alvarez,[22] could not be applied to the pig.

Alvarez's book,[22] with its extensive bibliography, is an important contribution to the literature of the mechanics of the digestive tract. It should be of especial interest to veterinary physiologists because of the fact that the animal mainly used by Alvarez in his experiments is the rabbit, a herbivorous animal. No review of this book can be given here. It may be remarked, however, that Alvarez has shown that stomach movements are far more complex than is usually described in the text-books. The methods giving best results in the hands of this worker are direct observations, multiple tracings, and motion-pictures of the stomach, in all cases exposed in a clear saline bath; and tracings from isolated segments.

Hunger Contractions

That there is a definite correlation between hunger and certain types of stomach contractions has been established by the work of a number of investigators. These movements and their relations have been extensively studied in small animals and man but only to a limited extent in the horse and apparently not at all in the pig. In the horse, Schalk and Amadon,[23] using the balloon method, found that hunger manifests itself in the form of powerful contractions that may begin as early as five hours after an ordinary meal, when, of course, the stomach still contains a good deal of food. Following the hunger periods there is an interval of quiescence varying from fifteen minutes to an hour or more in different animals.

Vomiting

Rodents, ruminants and solipeds seldom or never vomit, whereas carnivores and omnivores (except such as are rodents) vomit readily. Various views have been advanced to explain why certain animals do not vomit. The absence or rudimentary development of a nerve center of vomiting is held by some to be the chief cause. Hatcher and Weiss,[24] on the contrary, believe that the lack or poor development of a vomiting center is the effect, rather than the cause, of the absence of vomiting. The original purpose of vomiting was doubtless to assist animals in combating poisons taken in with the food, and these authors suggest that rats, and possibly other non-vomiting animals, owe their inability to vomit to the development of more effective means of combating poisoning, for example, selective absorption. Now nausea, which is experienced also by animals that do not vomit, would stop the ingestion of poisonous material, while selective absorption would delay or prohibit its entry into the blood. Hence vomiting would not occur. This condition, often repeated in successive generations, would lead eventually to inability to vomit because of long disuse of the vomiting center. In many animals vomiting still remains the chief means of combating poisoning. The mechanism of vomiting is comprehensively reviewed by Hatcher.[25]

References

[1]Bensley, R. R.: The Gastric Glands. Cowdry's Special Cytology (New York, 1928), I, p. 137.

[2]Lim, R. K. S., and Dott, N. W.: Observation on isolated pyloric segment and its secretion. Quar. Jour. Exp. Physiol., xiii (1922-23), p. 159.

[3]Bensley, R. R.: Cardiac glands of mammals. Amer. Jour. Anat., ii (1902), p. 105. Cardiac glands of mammalian stomach. Anat. Rec., iv (1910), p. 375.

[4]Ellenberger, W., and Scheunert, A.: Vergleichende Physiologie der Haussaugetiere (3rd ed.; Berlin, 1925), p. 236.

[5]Grosser, P.: Untersuchungen über den Magensaft. Zentralbl. f. Physiol., xix (1905), p. 265.

[6]Belgowski, J.: Beitrag zur Lehre von der Labmagensverdauung der Wiederkäuer. Arch. f. d. ges. Physiol., cxlviii (1912), p. 319.

[7]Karpov, L. V.: Digestion of vegetable and animal proteins by the gastric juice of the goose. Physiol. Abst., v (1920-21), p. 469.

[8]Rosemann, R.: Beiträge zur Physiologie der Vergauung. I. Mitteilung. Die Eigenschaften und die Zusammensetzung des durch Scheinfütterung gewonnenen Hundemagensaftes. Arch. f. d. ges. Physiol., cxviii (1907), p. 467.

[9]Haurowitz, F., and Petrou, W.: Abhandlung über Pankreasenzyme. Ztschr. Physiol. Chem., cxliv (1925), p. 68.

[10]Carlson, A. J.: Secretion of gastric juice in health and disease. Physiol. Rev., iii (1923), p. 1.

[11]Lim, R. K. S., Ivy, A. C., and McCarty, J. E.: Contributions to the physiology of gastric secretion. I. Gastric secretion by local stimulation. Quar. Jour. Exp. Physiol., xv (1925), p. 13: II. Intestinal phases of gastric secretion. Quar. Jour. Exp. Physiol., xv (1925), p. 55.

[12]Ivy, A. C., and Farrell, J. I.: Contributions to the physiology of gastric secretion. VIII. The proof of a humoral mechanism. A new procedure for the study of gastric physiology. Amer. Jour. Physiol., lxxiv (1925), p. 639.

[13]Ivy, A. C.: Newer physiology of the gastro-intestinal tract. Amer. Jour. Med. Sci., clxxiii (1927), p. 453.

[14]Lim, R. K. S., and Necheles, H.: Demonstration of a gastric secretory excitant in circulating blood by vividialysis. Proc. Soc. Exp. Biol. & Med., xxiv (1926), p. 197.

[15]Scheunert, A., and Schieblich, M.: Einfluss der Mikroorganismen auf die Vorgänge im Verdauungtraktus bei Herbivoren. Handbuch der normalen und pathologischen Physiologie (Berlin, 1927), III, p. 967.

[16]Scheunert, A.: Verdauung der Wirbeltiere. Oppenheimer's Handbuch der Biochemie (Jena, 1925), V, p. 56.

[17]See Sisson, S.: Some mechanical factors in digestion. Amer. Vet. Rev., xlv (1914), pp. 408, 513.
[18]Scheunert, A., and Kiok, F.: Zum Mechanismus der Magenverdauung beim Omnivoren. Arch. f. d. ges. Physiol., cxciii (1921), p. 16.
[19]Linton, R. G.: Animal Nutrition and Veterinary Dietetics (Edinburgh, 1927), p. 287.
[20]Bergman, H. D.: Personal communication.
[21]Cannon, W. B.: The Mechanical Factors of Digestion (London, 1911), p. 96.
[22]Alvarez, W. C.: The Mechanics of the Digestive Tract (2nd ed.; New York, 1928).
[23]Schalk, A. F., and Amadon, R. S.: Gastric motility studies in the goat and the horse. Jour. A. V. M. A., lix (1921), n. s. 12 (2), pp. 151-172.
[24]Hatcher, R. A., and Weiss, S.: Studies on vomiting. Jour. Pharmacol. & Exp. Therap., xxii (1923-24), p. 139.
[25]Hatcher, R. A.: The mechanism of vomiting. Physiol. Rev., iv (1924), p. 479.

Lice Not Killed by Drugs in Drinking Water

Drugs added to the drinking water will not remove lice from poultry. Officials of the Food and Drug Administration again proved this fact to the satisfaction of a jury in a case against the Reliance Manufacturing Company, at El Paso, Texas, recently. The jury affirmed the contentions of the government officials. The court imposed a fine and obtained a statement from the owner of the company that the manufacture of Reliance Lice and Mite Killer would be discontinued.

This is only one of a series of actions against this type of fraudulent product in which the courts have upheld the regulatory powers of the U. S. Department of Agriculture. A few years ago many such preparations appeared on the market and were tested thoroughly by W. S. Abbott, in charge of the entomological testing unit of the Food and Drug Administration, Dr. F. C. Bishopp, of the Bureau of Entomology, and their co-workers. All of these preparations were found to be of no value against lice and the market has been practically cleared of them.

The Reliance product, as determined by government chemists, was composed of commercial lime and sulphur solution diluted in three times its volume of water. The dose recommended was one teaspoonful of the solution in two gallons of drinking water. This dose, it was claimed, would remove lice, mites, blue bugs and other parasites from the poultry.

Dr. Abbott testified that his unit had made a complete practical test of the preparation, feeding it to lice-infested chickens as directed, and had found it positively worthless.

The theory advanced by the manufacturer of this preparation was that the sulphur would be deposited in and on the skin of the chicken and consequently would kill or drive away the parasites. This theory falls down, says Dr. H. E. Moskey, of the Food and Drug Administration, because sulphur is changed to sulphids in the intestinal tract and is eliminated as hydrogen sulphid. Moreover, chickens do not have sweat glands and do not eliminate waste products through the skin.

BACTERIOLOGICAL CONTROL OF CERTIFIED MILK*

By JOHN G. HARDENBERGH, *Plainsboro, N. J.*

Director of Laboratories, The Walker-Gordon Laboratory Company, Inc.

For a long time, bacteria counts have constituted one of the best standards we have for judging, at a distance, the sanitary conditions surrounding the production of fluid milk. Within recognized limits, these counts serve as a useful yardstick to measure the comparative excellence of different milks. Along with other standards, such as butter-fat content, we have developed the common practice of establishing bacteria-count restrictions for the various grades of milk. It must always be remembered, however, that official plate counts on milk represent only approximations that should not be overrated as to their accuracy.

As we pass up the scale in the grades of milk from poorest to best, bacteria counts within a grade have diminishing significance. Given two sources of supply that have attained reasonable perfection in the production and handling of milk, a variation simply in numbers of a few hundred, or even a few thousand, bacteria per unit can have little true meaning for the health officer or the consumer aside from some slight possible effect on keeping quality. Considering bacterial populations of milk from a numerical standpoint only, there can be little choice between a supply containing, say, twenty-five thousand per unit and one containing thirty or forty thousand, provided that conditions of production are essentially alike and equally good.

Hence, in the consideration of those grades of milk which stress a low germ-content, it becomes necessary to have some further means of evaluating their bacterial excellence. This can be done by determining the *types* or *kinds* of bacteria present, knowing that certain types are normal inhabitants of milk, whereas other types are abnormal and should not be present in appreciable numbers. Some progress in this direction has already been made by means of rather simple tests which permit the classification of predominant types as either lactic acid, peptonizing, putrefying, gas-producing, or combinations of these. Such grading is based on fermentation and curd tests, the results indicating, in a general

*Read before the Eighth New England Health Institute, Boston, April 14-19, 1930.

way, good quality, fair quality, bad or very bad, according to the predominance of types in the order above given. There are other tests, such as that for the colon-aerogenes group of bacteria, which are also of value in milk control work of this sort.

However, none of these differential tests are selective enough to be of great value in certified milk control, because this grade of milk is produced, not only under methods that insure a minimum of contamination with the ordinary, undesirable types of bacteria, but also under methods that are designed to eliminate harmful types as well. To my mind, one of the least important features of certified milk is the low germ-content itself. Today, many dairymen, with proper care, can and do produce milk that is rather consistently within the 10,000 count, but how many of them have requirements for the health of employes and of cattle, along with other refinements of production, that will insure the absence of pathogenic organisms?

In the light of modern knowledge of dairy science and in view of efforts to promote public health in every way possible, it is no longer sufficient for the producer, the health officer or the consumer to know merely that certified milk or milk of some other grade contains less than 10,000 bacteria per cubic centimeter: there must be assurance that every practical precaution has been taken to limit the possible presence of harmful types. The requirements for certified milk production already make this assurance reasonably certain; it is now our immediate problem to demonstrate how it can be made even more positive. If we know the diseases of man that occasionally, though rarely, are transmissible through milk, it should be possible to operate safety measures that will eliminate such potential sources of infection *at the source;* the same applies to diseases of dairy cattle. That is why the bacteriological control of certified milk today means more than supervision of total numbers. It means an agency for the application of preventive human and animal medicine in their modern forms.

What must be the nature of such an agency? As we see it, it takes definite form in the control laboratory, whose functions begin where medical and veterinary supervision leave off and, by supplementing the work of the doctor and the veterinarian, carries them to a purposeful and logical conclusion. After all, the problem of adequately safeguarding certified milk at the source is entirely feasible when the problem is resolved into its component parts. With respect to public health and the channels through

which milk may affect it, there are two phases that present themselves: first, bovine diseases directly transmissible and, second, human diseases directly or indirectly transmissible through milk. Leaving out of consideration those conditions that may influence the purity of milk only with exceeding rarity and which, therefore, cannot fairly be control problems, bovine diseases as we know them today are reducible to two items: tuberculosis and Brucella infection. In the same way, human diseases important to milk control work may be reduced to four items: typhoid fever, diphtheria, scarlet fever and septic sore throat.

Thanks to accomplishments in the control of bovine tuberculosis and in the recognition of carriers of typhoid and diphtheria organisms, these diseases may be cut from the list in so far as they relate to any new conception of the bacteriological control of certified milk. The tuberculin test of dairy herds is a dependable method of detecting tuberculous cattle, its efficiency being acknowledged as equal to that of any other biological test. As a result, certified milk herds are today, and have been for years, so free of tuberculosis that any chance of human infection with the bovine virus through certified milk may be regarded as nil. Much the same may be said for the human diseases—typhoid fever and diphtheria. Under the supervision of the medical officers of active medical milk commissions and with the present facilities for detecting carriers, the transmission of these diseases through certified milk can be and is prevented. To quote from a recent editorial[1] in the *New England Journal of Medicine:*

> Certified milk rarely comes, now, from tuberculous cattle; it is almost unheard of to have typhoid fever, scarlet fever or diphtheria occur as a result of its use.

This leaves, then, Brucella infection of dairy cattle and two human streptococcus infections, scarlet fever and septic sore throat, which merit special attention in the control of certified milk. Thanks again to the fundamental knowledge we have of these diseases, there are ways of combating all of them in so far as they constitute any real menace to a pure milk supply.

Brucella Infection and Certified Milk

During the past three years, there has been much agitation concerning abortion disease in dairy cattle and its relation to public health. Numerous articles have appeared in scientific and lay journals depicting various observations and conclusions as to either the perils of the infection, or, its apparent innocuous-

ness. In the resulting conflict of opinion, it has been extremely difficult to make any fair appraisal of the part played by milk in the etiology of undulant fever. Epidemiological studies have shown that, in a majority of cases, a history of consuming raw milk may be obtained but, in those same cases, fully fifty per cent had other possible contacts with *Brucella* infection. Deliberate attempts to produce human infection through the ingestion of milk heavily inoculated, either naturally or artificially, with *Brucella abortus* have failed conspicuously in at least two outstanding trials, whereas Brucella strains from swine and from goats are known to be much more pathogenic.

In certain sections of the country, *Brucella* infections in man are obviously milk-borne, while in others they are just as obviously due to occupational hazards in which there is direct contact with the infection, as, for instance, in slaughter-houses. In view of our unsettled knowledge of the disease in man and as long as milk is reputed to be a possible source of undulant fever infection, the supervision of certified milk supplies must provide adequate safeguards. The basic solution lies in the elimination of Brucella infections from the herds, which has already progressed with commendable rapidity. A number of certified herds are entirely free of the disease, many more are nearly free and, over the country as a whole, at least eighty-five per cent of the cattle in certified milk production are reported clean.

With respect to the elimination of abortion disease from dairy herds, it has been demonstrated that the agglutination test is reliable for the detection of *Brucella* infections. The removal of animals that fail to pass the test will serve to remove those that are shedding the organism in the milk. Exceptions to this are so rare as to have no practical significance. Occasional reports appear, and will probably continue to appear, that certain cows are found to eliminate *Brucella abortus* in their milk and yet fail to react to serum tests. On this basis, the agglutination test of cattle has been severely criticized as a means of protecting milk supplies. There is, in my opinion, no more reason for condemning this test than any other biological test. It is not claimed to be one hundred per cent efficient, yet it has very definitely accomplished its purpose with marked success, namely, the elimination of Brucella infections from milk and a satisfactory control of abortion disease in dairy herds.

Human Streptococcus Infections and Certified Milk

Scarlet fever and septic sore throat are of special interest in the bacteriological control of milk because they present possibilities in transmission fortunately not possessed by other infections. Both are due to specific strains of hemolytic streptococci which have been known, in a few instances, to establish themselves in the udders of dairy cattle. Recognition of this fact has explained the occurrence of certain milk-borne epidemics of scarlet fever and septic sore throat that were not understandable on the basis of direct human infection of the milk. This direct infection can occur but it is not reasonable to suppose that any epidemic would result, since it would be difficult for a human carrier to infect milk so grossly that any considerable portion would carry invasive quantities of bacteria. This is particularly true of large supplies wherein the ultimate dilution of any direct contamination might easily overcome the possibility of subsequent human infection.

Furthermore, Jones has shown in his studies of the influence of milk on the growth of scarlet-fever streptococci[4] and in his studies of the bactericidal properties of milk,[5] that certain streptococci of human origin are very susceptible to the effects of milk and that they fail to multiply in fresh unheated milk or in milk heated as high as 62°C. for 20 minutes. These findings would seem to indicate that no extensive epidemic of scarlet fever can occur by means of direct human infection of a milk supply.

However, serious invasion of milk supplies with human types of streptococci can occur through the medium of the cow as a secondary host. This has been the case in several milk-borne epidemics of septic sore throat in which *Streptococcus epidemicus* has been inoculated, as it were, into one or more quarters of an udder by an infected milker; the same occurrence has been noted in the case of *Streptococcus scarlatinae*. The subsequent multiplication of these bacteria in the medium furnished by the milk and udder tissues may result in large numbers of pathogens being discharged into the milk produced by the infected cow and so into the herd product, since there may be no immediate indication of resulting mastitis to warn the dairyman that the cow's milk should be discarded.

As a rule, in epidemics in which pathogenic streptococci of the human type have been traced to infected milk, we see the combination of a relatively small herd, perhaps only a few cows,

the milk from which is sold raw; the presence of a cow with an udder infected by a human carrier; methods of milk production which pay scant attention to udder disturbances and very little attention to the health of dairy employes. Under these conditions, it is possible for such a milk supply to carry infective doses of pathogenic organisms.

In large herds where the milk of any one cow may be mixed with the milk of, say, one hundred other cows, the dilution factor may operate to reduce the chances of infective doses of pathogens. We know that this must be the case because cows harboring *S. epidemicus* have been found at times when there was no occurrence of septic sore throat among consumers of the milk.

The significance of hemolytic streptococci in milk depends almost entirely upon whether the species present are of bovine origin, incapable of producing disease in man, or whether they are of human origin, having gained entrance to the milk directly from a human carrier or indirectly through the medium of a cow whose udder has been infected by a human carrier.

As far as hemolytic streptococci of bovine origin are concerned, it has been shown that they are often found in mixed herd milks, both raw and pasteurized. Several species have been identified, the most common being *Streptococcus mastitidis*, so named for its association with mastitis in cows. It may also be found living in the udders of cattle with no symptoms of udder inflammation. Its presence is not objectionable except as a possible indication of mastitis and it probably has no true significance to human health.

Our knowledge of the characteristics of pathogenic types of hemolytic streptococci has been put to the most practical use in the modern laboratory methods for control of certified milk production. Today it is a routine procedure on a few certified milk farms to examine on blood-agar the milk from every fresh and new cow prior to admission to the milking-line. In addition, the entire producing herds are checked at frequent (weekly) intervals by means of group samples which are also plated in blood-agar. The occurrence of any suspicious types of streptococci in such a group sample is followed up by a recheck of the individual cows contributing to the group in order to locate the animal responsible. These laboratory methods have been described in detail elsewhere.[2,3] They have been in routine operation for several years on a few certified milk farms and have given ample proof of their dependability.

At this point, objection must be made to statements that frequently occur in discussing the relation of bovine mastitis to human health. A recent health bulletin[6] says:

> Several epidemics of septic sore throat have been traced to mastitis, an udder infection of dairy cows.

A new book[7] recently off the press says:

> Inflammation of the udder, mastitis, commonly called "garget," may be caused by bacteria. If such condition exists, bacteria may be present in tremendous numbers in the milk, and be a potential source of septic sore throat among consumers.

Other similar citations may be found which give the casual reader entirely wrong impressions. Bovine mastitis, *per se*, can not cause scarlet fever or septic sore throat unless the causative organisms, *S. scarlatinae* or *S. epidemicus*, are present in the diseased udders. To the best of our knowledge, these disease-producing bacteria do not originate in the cow's udder but are transferred there by a human carrier and then only in exceptional cases. The importance of maintaining the distinction between human and bovine types of streptococci is obvious.

Pasteurization and the Control of Certified Milk

Up to this point, I have chosen to discuss the bacteriological control of certified milk from the standpoint of protection *at the source*, without reference to any processing method that might accomplish the same result.

I know that the control measures advocated for the protection of certified milk require considerable care and technical skill, but I submit that they represent fundamental principles in the production of the highest quality milk. It has been the aim, in producing certified milk, to remove the defects which make necessary the pasteurization of other grades of milk and it must be acknowledged that the effort has been substantially successful. On the other hand, we fully concur in the belief that pasteurization represents the cheapest and most efficient means of insuring the safety of fluid milk supplies that cannot be adequately protected by other means.

The growing sentiment in certain localities in favor of pasteurization of certified milk is quite evident. This sentiment may simply indicate the extreme precaution being taken in those localities to protect the public health. If those who favor pasteurization of certified milk will retain and maintain a firm and active interest in the principle of fundamentally sound milk at the source; if they do not relinquish in the least their efforts to

make every drop of certified milk fit and eligible for sale raw, even though part of it is to be pasteurized subsequently, then this action may be a step forward in milk hygiene. On any other basis it will be a step backwards, because the idea is becoming increasingly repugnant than any process can replace standards of health for dairy herds and dairy employes and standards of sanitation for dairy methods.

If the doctor, the health officer and the consumer desire the assurance of safety over and above the assurance rendered by technical supervision of production, then pasteurization of certified milk cannot be reasonably opposed. If the lack of pasteurization is a barrier which keeps people away from the use of and belief in a milk that has every possible care at the source, then this barrier should be removed by all means. Indeed, if care is taken not to lose sight of the basic principles and ideals of pure milk production, the pasteurization of certified milk may simply emphasize the progress that is being made in the promotion of preventive medicine and public health to which the sessions of this Health Institute are devoted.

REFERENCES

[1]Editorial: New England Jour. Med., ccii (1930), p. 385.
[2]Hardenbergh, J. G.: Laboratory control of certified milk on the farm. Proc. Internat. Asso. Dairy & Milk Insp., 1929.
[3]Hardenbergh, J. G.: Indentification and significance of hemolytic streptococci in milk. New England Jour. Med., ccii (1930), pp. 373-376.
[4]Jones, F. S.: Udder infection with streptococci of the scarlet fever type. III. The influence of milk on the growth of scarlet fever streptococci. Jour. Exp. Med., xliii (1928), 6, pp. 965-975.
[5]Jones, F. S.: Bactericidal property of milk. Certified Milk, iv (1929), 44, pp. 4-9.
[6]———: N. Y. State Dept. Health, Health News, vii (1930), 10, p. 40.
[7]Eckles, C. H., Combs, W. B., and Macy, Harold: Milk and Milk Products (The McGraw-Hill Book Co., New York), p. 98.

Commercial Firms Entertain Ladies

According to information received from Dr. C. L. Lehman, of Flanagan, Ill., the donations made by the following commercial firms made possible the entertainment of the ladies who were in attendance at the meeting of the Illinois State Veterinary Medical Association, held in Bloomington, July 8-9: Allied Laboratories, Ashe Lockhart, Inc., William Cooper & Nephews, Corn Belt Serum Company, Corn States Serum Company, Gregory Farm Laboratories, Haver-Glover Laboratories, Jensen-Salsbery Laboratories, Koen Laboratories, Norden Laboratories, North American Veterinarian, Regal Laboratories, Sharp & Smith, Shores-Mueller Company, Sutcliff & Case Company, Veterinary Medicine and Vitamineral Products Company.

BOVINE MASTITIS CAUSED BY STREPTOCOCCUS EPIDEMICUS*

By F. B. HADLEY, W. D. FROST, MILDRED GUMM *and* W. E. WELSH

From the Departments of Veterinary Science and Agricultural Bacteriology, College of Agriculture, University of Wisconsin Madison, Wis.

The dairyman is concerned with bovine mastitis because (1) it is often contagious, (2) it results either in temporary or permanent loss of udder function, and (3) it renders the milk unfit for use and so bars the product from trade channels. From the veterinary standpoint mastitis is now considered of as great importance as either tuberculosis or infectious abortion, the two other most prevalent and damaging infectious diseases of cattle. From the standpoint of human medicine this disease is of interest in that milk from infected cows may be the source of organisms responsible for septic sore throat and possibly some other diseases. Consequently, from any point of view, bovine mastitis is not only an important economic problem, but also one of public health importance.

The purpose of this paper is to present the results of recent studies on cows with both spontaneous and induced *Streptococcus epidemicus* (Davis) udder infections, rather than to discuss the history, occurrence, etiology, pathogenesis, symptoms, diagnosis, prognosis, treatment and prevention of bovine mastitis in general.

Considerable work has been done by a number of investigators in an endeavor to determine whether epidemics of septic sore throat are milk-borne. The epidemiologic studies leave little doubt that many outbreaks may reasonably be attributed to udder infections. In order to prove this assumption it would be necessary, first, experimentally to infect cows with *Streptococcus epidemicus* recovered from human cases of the infection and, second, to endeavor to infect volunteers with milk from cows known to be shedding the organism.

It is evident that the latter type of experiment is difficult, if not impossible, to arrange for. Accordingly we are left with only the former method of direct attack, although a series of monkeys are being exposed to different cultures of the organism.

*Presented before the joint meeting of the Chicago Medical Milk Commission and the Chicago Medical Society, Chicago, Ill., April 26, 1930.

Streptococcus epidemicus, the organism under consideration, is a member of the so-called beta-hemolytic streptococcus group. It forms a distinct, clear, wide zone of hemolysis on blood-agar, the colonies are large, and the surface colonies are watery, moist and spreading. Many of the colonies either go to the surface of the agar or the bottom of the plate. It has a high hemolytic titre, possesses a capsule as shown by the moist India ink method, fails to hydrolyze sodium hippurate, is a low acid-producer in dextrose, forms acid in lactose, saccharose and salicin, and fails to ferment mannite.

Spontaneous Mastitis Caused by Streptococcus Epidemicus

A recent paper by Frost, Thomas, Gumm and Hadley,[1] on seventeen cows demonstrated to be shedding *Streptococcus epidemicus* in their milk, indicates that this organism appears to be rather widely distributed in cows, so must occasionally contaminate milk and other dairy products from time to time. However, it is significant that only two of the seventeen cows reported (both in one herd) were associated with an epidemic of septic sore throat, while the milk from the fifteen others did not, as far as could be determined, cause disturbances in persons who consumed it.

Since the above-mentioned paper was released, we have isolated a very similar, if not identical, streptococcus from a cow in a herd at Baraboo, Wisconsin. The same organism was recovered from the throats of a number of persons in that city suffering from septic sore throat. One of the cultures used to infect some of the experiment cows, to be referred to subsequently, was recovered from an ear abscess of a Baraboo girl and designated as the IJ culture. This organism was virulent enough to cause the death of mice given 0.5 cc of a broth culture within 24 hours. The other culture was from the Baraboo cow designated as 18 in table I. It was found to be pathogenic for white mice, but not for a dog, guinea pig, or pigeon.

Cow 18 had a fulminating attack of mastitis in the left fore quarter, which had dried up before she was detected. The period during which this infection lasted was such as to account for the onset and spread of the epidemic. Later we were able to express from this quarter a few drops of pus, which contained *Bacillus pyogenes*, an organism that we regarded as a secondary invader. Later the other fore quarter became infected and we were able to study the flora of this quarter over a period of three

months. The etiological factor was *Streptococcus epidemcius*, but it was never found in large numbers.

This makes three cows suffering from an infection with *Streptococcus epidemicus* and connected with epidemics of septic sore throat which we have had the opportunity of studying; two were reported on in detail in the article by Frost, Thomas, Gumm and Hadley, and the above cow will be reported on in greater detail later.

The second group of cows, namely, those infected with *Streptococcus epidemicus*, but not associated with clinical disease in man, has been added to by the incidental discovery of five other cows, bringing the total to twenty. One of these cows was found in a very small herd near Madison and was disposed of before any thorough study could be made. She is cow 19 in table I.

The four others were in another farmer's herd of twenty milking cows, also near Madison. They are designated as cows 20, 21, 22 and 23 in table I. Detailed records have been kept on these cows but are not included because they would require too much space. It should be stated that cow 20 rapidly lost weight, although continuing to produce about 40 pounds of milk daily and, like the others, had a decided rise in temperature. It is significant that the first case of mastitis in this herd occurred in the right fore quarter of cow 22, following a teat injury from being stepped on. She became a "hard milker," so a wax teat-dilator was used to break down adhesions that had formed in the teat sinus. On April 8, the milk from this quarter was found to carry 12,000,000 typical *Streptococcus epidemicus* organisms per cc; on April 14, the count was 3,400; and on April 23, it was 60,000. It seems reasonable to believe that the dilator became contaminated with *Streptococcus epidemicus* from the throat of the milker who inserted it twice daily during the ten days or two weeks it was employed. However, this hypothesis has not been definitely established; in fact, throat swabs from the owner and his wife were negative, but no swabs were taken from the hired man.

Arrangements have been made to keep three of these cows under observation for at least a year, to secure information relative to the course of the infection and the damage it produces in the udder. Moreover, it is important to learn whether such cows cease to shed the organism and, if they do, whether they may be returned to the milking line with safety. The question of recrudescence of the infection is also of interest in this regard.

TABLE I—*Records of cows whose milk contained Streptococcus epidemicus (Davis)*

COW	SPECIAL DESIGNATION	MONTH DISCOVERED	SIZE OF HERD (HEAD)	TIME UNDER OBSERVATION	TESTS		STREPTOCOCCUS EPIDEMICUS COUNTS		QUARTERS INFECTED	CONDITION OF MILK
					TOTAL	POSITIVE	MINIMUM	MAXIMUM		
18*	Baraboo	December	29	3 months	6	6	1,040	5,600	Right fore	Abnormal
19	Fridal	December	20	1 day	1	1	?	?	?	Abnormal
20	Up. 1	April	20	21 days	3	3	900,000	24,000,000	Right hind	Abnormal
21	Up. 2	April	20	21 days	3	3	48,000	6,800,000	Left fore	Abnormal
22	Up. 3	April	20	21 days	3	3	3,400	12,000,000	Right fore	Abnormal
23	Up. 4	April	20	7 days	2	2	120,000,000	600,000,000	Right hind and fore	Abnormal

*Associated with an epidemic of sore throat.

A summary of the records on the six cows referred to above is presented in table I, which is an extension of table I in the paper already cited.

Induced (Experimental) Mastitis Caused Streptococcus Epidemicus

Davis and Capps[2] have reported that they were unable to induce mastitis in a cow simply by smearing cultures of hemolytic streptococci of human origin on the uninjured surfaces of the teat of the cow. They did succeed, however, in infecting a cow's udder by abrading the skin at the lower end of the teat and applying a culture of the organism, but the milk never became gargety. They also established a mastitis in another cow by introducing the culture into the teat-canal, but the organisms disappeared from the milk within a month and the udder became normal after about six weeks.

Mathers[3] carried out a somewhat similar series of experiments using in one cow a culture of a hemolytic streptococcus that Davis had isolated from the udder of a cow with mastitis; a culture of the non-pathogenic, non-hemolytic *Streptococcus lacticus;* and a culture of a strain of streptococcus isolated from a fatal case of human streptococcal peritonitis following an attack of septic sore throat during the Chicago milk epidemic in 1912. From his work and that of Davis and Capps, he and Davis concluded that the human types of streptococci, when inoculated into cows, are uniformly more virulent for the cow than are the streptococci derived from bovine sources.

On its face this conclusion seems illogical, as one would suppose that pathogenic microörganisms recovered from a given species of animal would be more highly virulent for other members of that species than for those of an entirely different and quite widely separated species.

In order to learn whether strains of the *Streptococcus epidemicus* of both human and bovine origin are pathogenic for cows and capable of inducing mastitis, the three experiments now to be described were conducted. Blood-agar slant growths of the organisms, about 24 hours old, were used for infecting the animals. Blood-agar plates were used to determine the bacterial counts of the milk.

As the cow's mammary glands, commonly spoken of as the udder, are unique anatomically, it may be well to mention their features. The udder is considered as consisting of two glands,

divisible into four quarters. Although the halves of the udder are separated by a median septum, there is no visible division between the two quarters of the same side. However, injections of fluids of different colors into the two teats of the same side demonstrate that the cavities drained by them do not communicate.

Experiment 1: Three normal lactating cows demonstrated to have no beta hemolytic streptococci in their milk were selected. Each cow had been in the milking-line of a certified dairy but was approaching the end of her lactation period, although still giving considerable milk. The side of the right fore teat of each was scarified, inoculated, and then covered with a thin coating of "new-skin." Cow 1 was inoculated with the culture from the Baraboo cow already referred to; cow 2 received the IJ culture; cow 3 the culture 76 from a human case occurring in the Lee, Massachusetts, septic sore throat epidemic in June, 1928. These animals were kept under close observation for 16 days but never showed a rise in temperature or disturbed appetite. No evidence of garget was shown by either the strip cup or bacteriologic examination of the milk in cows 1 and 3. Cow 2, however, on the ninth, tenth and eleventh days after inoculation, showed small yellow clots on the gauze of the strip cup in the milk from her right fore quarter. As this milk and that from all other quarters of this cow was at all times free from beta hemolytic streptococci, the transient mastitis was attributed to some factor other than the inoculum. It was concluded that the "new-skin" preparation might have exerted a germicidal action on the organisms, so no conclusions were drawn from the experiment.

Experiment 2: As none of the three cows used in experiment 1 reacted, we decided that they were suitable for use in experiment 2. The technic of inoculation in this series of tests was varied by simply smearing the inoculum on the tips of all four teats of each cow with a swab, and twirling the swab, but not inserting it in the meatus of the teat. The three different strains formerly used were employed, but with this difference: Cow 2 was inoculated with the Lee culture and cow 3 with the IJ culture, while cow 1 received the same culture which with she had been inoculated, *viz.*, the Baraboo cow culture. The results in cow 1 were practically negative, although the day following the inoculation the milk from her left front quarter on culture revealed 8,000 beta streptococci per cc but none thereafter. On the third and

fourth days after exposure, the strip cup was positive and a slight sensitiveness developed in this quarter, but these signs then disappeared and did not recur. Cow 2 never showed the slightest sign of a reaction. The record of cow 3, however, is extremely interesting. Not only did she exhibit a rise of temperature up to 103.8° F. and develop an acute mastitis, but also a beta hemolytic streptococcic count that reached a maximum of 120,000,000 per cc in her left hind quarter. The count gradually decreased to 6,000 per cc, but likely would have increased from this minimum if the cow had been kept under observation longer. This mastitis persisted for one month or until she was disposed of. The milk was thick in consistency and yellow in color from the day following inoculation to the end.

The results secured from experiment cow 3 justify the conclusion that it is possible to establish an acute, persistent bovine mastitis with *Streptococcus epidemicus* (Davis) of human origin by simply smearing the organisms on the end of the teat. They also support the opinion held by others that the human type of hemolytic streptococci is relatively more virulent for cows than is the alpha hemolytic type which, according to Carpenter,[4] as well as Hardenbergh and Schlotthauer,[5] is the most common cause of infectious bovine mastitis.

Experiment 3: This experiment was undertaken to determine whether cows 1 and 2, which had been shown in the two previous experiments to possess some resistance to exposure with *Streptococcus epidemicus*, would react to a more severe method of exposure. Accordingly, the right front and left rear teats of each were scarified completely around the meatus and smeared with a 24-hour blood-agar culture of the organism. Cow 1 was inoculated with the Baraboo cow culture; cow 2 with the Lee, Massachusetts, culture. The result in the latter was entirely negative. The result in cow 1 also was negative except that the day following the inoculation, milk from the two inoculated quarters showed 320 and 600 beta hemolytic streptococci per cc respectively. These two cows were kept under observation for two weeks, but as the organism was not isolated again and they did not develop clinical symptoms, they were then sold for slaughter.

The thought suggests itself that the Lee culture, which is now two years old, has lost much of its original virulence. It also appears that the bovine culture used in these experiments was not so virulent as would have been reasonable to believe, con-

sidering its source, age and pathogenicity for the cow from which it was originally recovered.

Experiment 4: This experiment was planned to secure more evidence relative to the transmissibility of *Streptococcus epidemicus* to cows. The test animals consisted of four normal lactating cows comparable in every way to those used in experiments 1, 2 and 3. They were designated as 4, 5, 6 and 7, and were placed on trial April 22. The method of exposure was identical with that used in experiment 2. The inoculum used was the IJ culture exclusively.

Cow 4 was kept under observation for one month but never gave evidence of a reaction. Cow 5 developed an infection in her right fore quarter 60 hours after exposure; her temperature reached a maximum of 103.8° F.; beta hemolytic streptococci were demonstrable, however, for only two days; the milk became thin and contained slimy clots. Her right rear quarter showed a few hundred hemolytic streptococci the day after exposure, none on the two following days, but a few on the next three days. Cow 6 showed a few betas on the second day but none thereafter.

Cow 7 was the most interesting because she was shown to be highly susceptible. Forty hours after exposure, she had developed a marked mastitis in the left and right hind quarters and 60 hours after in the right fore. Her temperature reached a maximum of 105.6° F. A sudden marked drop occurred in the amount of milk produced as well as in its appearance; the leucocyte count of the milk by the Stokes method increased from a normal of about 10 to a maximum of 260; the pH value of the milk dropped from a normal of about 6.7 to 6.0. The left hind quarter showed beta hemolytic streptococci for four days, but never thereafter. The right hind quarter shed enormous numbers of betas for about four weeks, but has not since, although the brom-cresol-purple test shows that there is still evidence of mastitis in this quarter. The bacteria count of the milk from the right fore quarter went as high as 2,160,000,000 betas per cc. They persisted in this quarter over a period of two months. The left fore quarter never reacted in any way.

Experiment 5: The purpose of experiment 5 was to determine whether immunity had been established in any of the quarters of susceptible cow 7. It will be recalled that this cow did not develop an infection in the left fore quarter from the first exposure; that she developed only a very temporary infection in the left hind, but that she did develop a marked persistent infection

in both the right fore and hind quarters. Upon re-exposure with the same culture which was used in experiment 4, the interesting observation was the development of a marked mastitis in the left hind quarter, which appears to be as severe as that produced after the first exposure in two of her other quarters, and which has persisted for 10 days and is likely to much longer. The left fore quarter, however, did not respond.

This proves that cows infected with *S. epidemicus* in one quarter do not necessarily establish an immunity towards this organism in other quarters; and moreover, something that is even more interesting, one attack of the infection in one quarter does not establish an immunity against subsequent attacks. It shows also that some quarters of susceptible cows must possess a very effective natural immunity, while other quarters are quite vulnerable.

General Discussion

Because of their importance and newness, certain features already discussed should be repeated for the sake of emphasis. Up to the summer of 1925, when the work of Brown, Frost and Shaw[6] was done, no cows infected with *Streptococcus epidemicus* and not associated with septic sore throat had been reported. We now have a record of twenty such infected cows. Eight of these were found in the certified herds where a systematic search is made for them, six from one farm, and one each from two other farms; twelve of them have been incidentally found by the examination of milk samples sent in by veterinarians because of the serious and persistent character of the mastitis. These interepidemic cases of epidemicus infection in cows have counterparts in the cases reported by Pilot and Davis[7] as having been discovered in man.

A very momentous question arises in this connection, and that is whether or not the organism causing these interepidemic infections is the same as that which produces septic sore throat in man. They are identical as far as all our laboratory tests and animal experiments show. An attempt is being made to throw further light on this question by experiments on monkeys, but the results so far secured tend to the conclusion that monkeys are not susceptible to this organism.

Our work on induced or experimental infections seems to show that a teat or udder trauma is not necessary for infection, as was formerly supposed. It would naturally follow from this that the opportunity for cows to acquire the disease from infected

milkers is much more likely to occur than we had believed was possible.

The question of acquired immunity is not by any means settled by these experiments, but it has been shown that cows may have a decided natural immunity in some quarters, yet have a marked susceptibility in others; furthermore, that one attack of the infection does not protect against subsequent attacks.

Ample evidence is presented to conclude that while the appearance of the milk cannot be taken as a guide in determining whether it is contaminated with pathogenic streptococci, that the strip cup, leucocyte counts, brom-cresol-purple, and other color indicators for the determination of the pH value are valuable for this purpose, but that a bacteriologic examination of the milk is the only reliable means of diagnosis. Furthermore, it is justifiable to conclude that a very acute mastitis may be caused in cows by infection with *Streptococcus epidemicus* of human origin. Further observations should show the extent and persistence of the damage. There seems to be no doubt of its contagiousness, however.

ACKNOWLEDGMENT

This work was aided by a grant from the American Association of Medical Milk Commissions, and the normal cows for the experiment were furnished by Howard T. Greene, Brook Hill Farms, Genesee Depot, Wisconsin.

REFERENCES

[1]Frost, Thomas, Gumm and Hadley: Jour. Inf. Dis., xlvi (1930), p. 240.
[2]Davis and Capps: Jour. Inf. Dis., xv (1914), p. 135.
[3]Mathers: Jour. Inf. Dis., xix (1916), p. 222.
[4]Carpenter: Jour. A. V. M. A., lxvii (1925), n. s. 20 (1), pp. 317-323.
[5]Hardenbergh and Schlotthauer: Jour. Inf. Dis., xl (1927), p. 667.
[6]Brown, Frost and Shaw: Jour. Inf. Dis., xxxviii (1926), p. 381.
[7]Pilot and Davis: In press.

Commencement

At the sixth annual summer school commencement held at the Kansas State Agricultural College, July 31, 1930, the degree of Doctor of Veterinary Medicine was conferred upon two candidates: Wesley Watson Bertz (high honors) and Andy Crawford.

At the same time the degree of Master of Science was conferred upon two veterinarians: Dr. John Flower Bullard (Corn. '22), of West Lafayette, Ind., and Elden Emanuel Leasure (K. S. A. C. '23), of Manhattan, Kans. Dr. Bullard's thesis was entitled "The Effects of Feeding Various Members of the Colon-Typhoid-Enteritidis Group to Anemic and Non-Anemic Pigs," and Dr. Leasure's subject was "Sweet Clover Disease Investigation."

MONTHLY TREATMENT OF GOATS WITH TETRACHLORETHYLENE

By E. A. TUNNICLIFF,* *Sonora, Texas*

Veterinarian, Substation No. 14, Texas Agricultural Experiment Station

Forced grazing over infested ranges year after year favors the development of intestinal parasites. In many cases it is necessary to treat for stomach worms two to six times a year. The most progressive ranchers often treat their animals every sixty days with the exception of a few cooler months in the winter. Following the introduction of tetrachlorethylene for intestinal roundworms, repeated treatments prompted questions regarding its effect upon the parenchymatous organs. Stated in another way, could tetrachlorethylene be repeatedly administered in therapeutic doses without injury to the host? Shortly after experiments designed to supply such information had been started at this station, Schlingman[1] reported that sheep treated for one year at intervals of 30 days showed no apparent harmful effects from the drug. Lamson, Robbins and Ward[2] worked with dogs, puppies, cats, mice and guinea pigs to conclude that "no appreciable pathological and functional changes" result.

HANDLING OF ANIMALS

This investigation has been confined to Angora and Spanish bucks, chevons and does of different ages. Their weights varied from 30 to 150 pounds. The animals were pastured on the same range throughout the entire test of 14 months, during which time monthly therapeutic doses of tetrachlorethylene were administered. No attempt was made to withhold feed or water before or after treatment. To observe any immediate ill effects which might have been produced, the animals were held under observation for a few hours after each treatment, in a barren corral where an adequate water supply was available.

Beginning after the fourth successive monthly treatment, a goat was autopsied and examined for gross pathological changes on the second, third, seventh and fourteenth days after treatment, until all but six females had been sacrificed. There were two exceptions to this plan. First, an animal was not autopsied

Received for publication, May 5, 1930.
*Resigned, January 1, 1930.

on the seventh day after the seventh treatment. Second, the autopsy examinations were concluded at the end of the ninth treatment and without examination of the goat which should have been killed 14 days after this regular monthly treatment. At this time it seemed inadvisable to sacrifice the six remaining females. Therefore, the treatments were continued on through the fourteenth month to determine if clinical illness might be detected.

TABLE I—*Goats treated on successive months with tetrachlorethylene.*

GOAT	TREATMENTS	AUTOPSIED (DAYS AFTER LAST TREATMENT)	MACROSCOPIC LESIONS	INTERNAL PARASITES
361 Buck	4	2	Atrophy of myocardium.* Liver—friable. Small intestine—catarrhal	Cecum—a few cecal worms
363 Buck	4	3	Negative	None
371 Buck	4	7	Negative	None
378 Buck	4	14	Pericardium—small amount of clear fluid	None
377 Buck	5	2	Negative	None
369 Buck	5	3	Small intestine—slightly catarrhal	Abomasum—a few stomach worms
365 Buck	5	7	Slight atrophy of myocardium	None
375 Buck	5	14	Slight atrophy of myocardium	None
367 Buck	6	2	Small intestine—catarrhal	Abomasum—5 dead stomach worms
374 Buck	6	3	Negative	None
348 Chevon	6	7	Negative	None
349 Chevon	6	14	Negative	Abomasum—several stomach worms
350 Chevon	7	2	Negative	Abomasum—several stomach worms
351 Chevon	7	3	Small intestine—catarrhal	Abomasum—several stomach worms Colon—several nodular worms
339 Doe	7	14	Negative	None
370 Buck	8	2	Negative	None
376 Buck	8	3	Negative	Colon—several nodular worms
347 Chevon	8	7	Negative	Abomasum—several stomach worms
362 Buck	8	14	Negative	None
360 Buck	9	2	Negative	None
352 Buck	9	3	Negative	None
372 Buck	9	7	Negative	None

*Difficult urination for 2 months previous to autopsy, may have been responsible for lesions.

Treatment and Result

The first capsules were given in August, 1928. The therapeutic dose, of a 5-cc tetrachlorethylene capsule, was repeated each succeeding month on all surviving animals. At autopsy a macroscopic examination was made of all organs for pathological changes. The abomasum and intestinal tube were observed for internal parasites. The results from each animal are recorded in table I.

Specimens 361, 363, 371 and 378 received 4 successive monthly treatments. Buck 361, autopsied two days after the fourth treatment, showed atrophy of the myocardium, friable liver, and catarrhal inflammation of the small intestine. A few cecal worms were found in the cecum. As previously pointed out, this goat had not been well for two months prior to autopsy. Difficult urination had been observed and upon postmortem examination lesions were found which may have been produced by this disturbance. Therefore, the heart and liver changes should not be positively attributed to the effect of this drug without confirmatory tests. No gross lesions and no intestinal parasites could be found in bucks 363 and 371, autopsied 3 and 7 days after the fourth treatment. A small quantity of clear fluid was present in the pericardium of buck 378, examined 14 days after this treatment. No intestinal parasites were present in these three goats.

Specimens 377, 369, 365 and 375 received 5 successive monthly treatments. The organs of buck 377, examined 2 days after the last treatment, were normal. No intestinal parasites were found. Buck 369, examined 3 days after the last treatment, revealed a slight catarrhal inflammation of the small intestinal mucosa and a few stomach worms in the abomasum. A slight atrophy of the myocardium was found in bucks 365 and 375, autopsied 7 and 14 days after the last treatment. No intestinal parasites were present in either animal.

Specimens 367, 374, 348 and 349 were autopsied 2, 3, 7 and 14 days respectively after the sixth monthly treatment. The small intestine of buck 367 showed a slight catarrhal inflammation. Five dead stomach worms were present in the abomasum. No visible lesions were found in the three other goats of this series. Buck 374 and chevon 348 were free of intestinal parasites, but chevon 349 had several stomach worms in the abomasum.

Specimens 350, 351 and 339, autopsied 2, 3 and 14 days respectively after the seventh monthly treatment, showed no patho-

TABLE II—*Goats treated for fourteen successive months with tetrachlorethylene*

GOAT	DATE OF TREATMENT													RESULT
	1928					1929								
	8–7	9–7	10–7	11–20	12–28	1–18	2–18	3–18	4–24	5–20	6–29	7–25	8–19	6–26	
338 Doe*	3cc	5cc	5cc	5cc	5cc	5cc	5cc	5cc	5cc	5cc	5cc	5cc	5cc	5cc	2– 3–29, kidded normally 11–26–29, physical condition excellent
340 Doe*	3cc	5cc	5cc	5cc	5cc	5cc	5cc	5cc	5cc	5cc	5cc	5cc	5cc	5cc	2– 4–29, kidded normally 11–26–29, physical condition excellent
341 Doe*	3cc	5cc	5cc	5cc	5cc	5cc	5cc	5cc	5cc	5cc	5cc	5cc	5cc	5cc	2–10–29, kidded normally 11–26–29, physical condition excellent
342 Doe*	3cc	5cc	5cc	5cc	5cc	5cc	5cc	5cc	5cc	5cc	5cc	5cc	5cc	5cc	2–12–29, kidded normally 11–26–29, physical condition excellent
343 Doe*	3cc	5cc	5cc	5cc	5cc	5cc	5cc	5cc	5cc	5cc	5cc	5cc	5cc	5cc	3–14–29, kidded normally 11–26–29, physical condition excellent
354 Doe	5cc	5cc	5cc	5cc	5cc	5cc	5cc	5cc	5cc	5cc	5cc	5cc	5cc	5cc	2– 8–29, kidded normally 11–26–29, physical condition excellent

*Small Spanish does. First treatment, small capsules.

logical lesions other than a catarrhal inflammation of the small intestinal mucosa in chevon 351. Several stomach worms were present in the abomasum of chevons 350 and 351. Nodular worms also were present in chevon 351. No intestinal parasites were found in doe 339. The goat which should have been examined on the seventh day after treatment was not autopsied.

Specimens 370, 376, 347 and 362, autopsied 2, 3, 7 and 14 days respectively after the eighth monthly treatment had been completed, showed no pathological lesions in any of the organs examined. No intestinal worms were found in bucks 370 and 362. Several stomach worms were found in the abomasum of chevon 347.

Specimens 360, 352 and 372, examined 2, 3 and 7 days respectively after the ninth monthly treatment had been administered, revealed no gross lesions or intestinal parasites. A fourteen-day animal could not be autopsied without sacrificing one of the treated females. Since additional data were desired from these does, the postmortem examinations were concluded without completing this last autopsy series.

The six females shown in table II were carried on through the fourteenth successive monthly treatment to observe the effect of the treatments. Spanish does 338, 340, 341, 342 and 343, weighing about 30 pounds at the time of the first treatment, August 7, 1928, were given 3-cc capsules, but thereafter the 5-cc capsules were administered as with the other goats. The five Spanish does and one Angora doe had received therapeutic doses of the drug through the entire gestation cycle. These females were alive and all in good physical condition at the time the tests were closed, November 26, 1929.

Summary

Several of the goats showed changes such as slight atrophy of the myocardium and mild enteritis. The majority of the organs were normal in every way.

The six does passed through normal gestation cycles after 7 to 8 successive monthly treatments with tetrachlorethylene. They remained in excellent condition after fourteen successive monthly doses of the drug.

Conclusion

The results with these two groups of goats suggest that tetrachlorethylene, when repeatedly administered in therapeutic

doses, is not toxic. It does not possess objectionable features which could be detected by macroscopic examination of the internal organs or by the physical appearance of the animals.

REFERENCES

[1]Schlingman, A. S.: Further miscellaneous tests of tetrachlorethylene as an anthelmintic. Jour. A. V. M. A., lxxv (1929), n. s. 28 (1), pp. 74-85.

[2]Lamson, P. D., Robbins, B. H., and Ward, C. B.: The pharmacology and toxicology of tetrachlorethylene. Amer. Jour. Hyg., ix (1929), pp. 430-444.

Horses Play Important Role in Quaker City

Dr. Ward Giltner, of Michigan State College, forwarded the following abstract of an article which appeared in a recent issue of *Municipal Sanitation*, under the title, "Disposal of Philadelphia's Street Wastes, Ashes and Garbage a Complex Job."

More than $1,000,000 goes for supplies, material and live stock. It costs $115,000 a year to shoe the 1,500 horses and about $170,000 for repairs, maintenance and hiring of motor vehicles. The cost of maintenance per horse per day works out at something like $1.75. This includes superintendence, foremen and stable labor, veterinary service, medicine, shoeing, feed, stable repairs and rentals, harness repairs, light and power.

There are more than a dozen stables, only one of which is owned by the city. Stable rental is about $11,000 a month, and the city has had to reconstruct most of the stables, refit them, and, of course, must keep them in repair. Inspection shows them to be well fitted and well maintained, with unusually good sanitary conditions, sick bays and every convenience.

The annual rent bill of the Bureau is about $130,000.

Each stable is visited daily by a veterinarian and there is an infirmary at Model Farm, where a sick animal may be restored to health. The Bureau maintains a complete wagon shop in which it builds its own wagons and repairs them. It also maintains a garage and repair shop for its trucks, and the forty-odd flushing machines used to wash off paved streets. A wagon is more easily and cheaply loaded than a truck, and much more economical in house-to-house collection, except where distance between houses is considerable, as in some outlying sections. The horse soon becomes well trained and needs hardly any direction, stopping, starting and even turning, and, on the whole, acting as an intelligent helper to the men who do the collecting.

PERSONALS

Dr. E. T. Hallman (A. P. I. '10) left East Lansing, Mich., August 2, accompanied by his family, on an automobile trip that would take them as far west as Los Angeles, where they planned to attend the A. V. M. A. convention. Dr. Hallman's itinerary included numerous stops at veterinary colleges laboratories and other institutions.

Dr. A. L. Warth (West. '08), of Garnett, Kans., reports that on August 2, his automobile was stolen from in front of his office at 122 5th Ave. The car was insured, but unfortunately for Dr. Warth, at the time it was stolen, the car contained about $350.00 worth of instruments and equipment which were not covered by the insurance. Dr. Warth suggests that veterinarians profit by his experience.

THE FUNCTIONS OF LIVE STOCK SANITARY OFFICIALS IN THE TUBERCULOSIS OFFENSIVE*

By C. C. HISEL, *Oklahoma City, Okla.*

State Veterinarian of Oklahoma

The functions of live stock sanitary officials in the tuberculosis offensive began in 1917, with the United States Bureau of Animal Industry cooperating by securing a contract with any cattle-owners interested in cleaning up their herds on what was known as the Accredited Herd Plan, which plan was inaugurated for the purpose of demonstrating that herds could be cleaned up and kept clean.

This plan was followed by all the states and the Bureau of Animal Industry until 1922, when the real eradication program started with a cooperative agreement between county, state and federal governments. Up to 1922, the work was considered educational in the true sense of the word, but complete eradication would have been impossible under the plan with the herd as a unit.

The state representatives in this conference met at Texarkana, on August 30, 1927, and organized the Southwestern Tuberculosis Conference, with the following states participating: Arkansas, Louisiana, Mississippi, Texas and Oklahoma. The sanitary officials of the various states and the entire medical profession have waged the fight against tuberculosis as one unit for the good of the people and the live stock industry. Tuberculosis has cost more human lives until recently than any other disease. At the same time, it has caused the greatest economic loss to the live stock industry, save that of contagious abortion alone.

As a direct result of the educational work done by this conference, Oklahoma and Texas have secured much needed legislation which has resulted in Dallas County, Texas, and Canadian County, Oklahoma, being declared modified accredited free areas. There can be no doubt that it will serve Arkansas advantageously, as well as Louisiana and Mississippi, especially by the time the conference meetings are scheduled for these states.

Perhaps the next most important problem is to secure a greater number of competent veterinarians for the purpose of testing cattle at a more rapid rate than heretofore. At the same time,

*Presented at the Southwestern Tuberculosis Conference, Little Rock, Ark., May 7, 1930.

we must be able to sell the state legislatures on the importance of complete eradication, in order that funds for expense and indemnity may be sufficiently liberal, especially in case of indemnity. Unless a liberal indemnity is paid the owners of tuberculous cattle, they will not cooperate as they should, if at all. Since the eradication of tuberculosis is done for the good of society as a whole, it is right and proper that the public as a whole should participate in the loss of the animals. Although it is a well known fact that this disease is transmitted to the human family through dairy products, which are essential for the growth and development of the oncoming generations, nevertheless, quality dairy products are now being demanded more than ever before, and as time goes on this demand will become more and more pronounced.

The Public Must Help

We who are officials are eager to have the assistance of the general public in this the greatest sanitary project in the civilized world, realizing, as we do, that the laity as a whole can do much by helping to secure state funds with which to work more hastily and effectively. On the other hand, we must not let politics enter in and disrupt a program where human lives are at stake each day. If we can convince the people as a whole that they owe their families milk and other dairy products which are free of the bacterium of tuberculosis, the greatest victory over the loss of life will be won, and at the same time untold millions saved for the live stock industry. As public officials and health workers we owe this service to our great nation.

The situation that developed in Chicago has been described by Dr. J. E. Gibson, B. A. I. inspector in charge of tuberculosis eradication in Indiana, as follows:

> "Procrastination is dangerous in any event and is particularly dangerous when applied to disease, whether it be disease of the human or of the lower animals. No better example of the disastrous results of procrastination could be cited than to point out to you what took place in that great dairy district known as the Chicago milk-shed. For years the dairymen of that district had stood out against the tuberculosis eradication program. They knew, and publicly admitted, that their dairy cows were diseased and yet they refused to submit them to a tuberculin test and demanded the right to ram the milk from their tuberculous cows down the throats of Chicago's innocent and helpless children until the situation had come to be a public scandal. And then there rose up a great public benefactor, in the person of Dr. Bundeson, then Commissioner of Health, who served notice on these dairymen that on and after April 1, 1926, no milk should be sold in the city of Chicago to be used for human consumption unless it came from cows which had been tuberculin tested under state and federal supervision. It was then that there was hurrying to and fro and

the authorities were beseeched from every direction to apply the test and as a result, many entire herds were completely wiped out. During the month of March, 1926, 37,000 dairy cows passed through the Chicago stock-yards on their way to the shambles and scores of dairymen were brought to the brink of utter financial ruin, all because they had failed to begin in time to rid their herds of a disease that is not self-limiting and will not eradicate itself but which will, upon the other hand, continue to spread after once introduced into the herd until the entire herd becomes diseased to a greater or less degree."

The fight against animal tuberculosis is being waged by 44 of the 48 states at the present time, with no signs of letting up until the last tuberculous animal has been disposed of, according to a specific plan laid down by the live stock sanitary officials and supported by state statutes.

The conference here assembled is entitled to the whole-hearted support of the rich and poor, white and black, educated and uneducated alike. The educated should be held responsible for the education of the uninformed until this great victory is won. The rich among us should aid and assist communities which will find it hard to raise funds by taxation on an already tax-ridden agriculture. In other words, in this fight, it is a good time for some of our multimillionaires, for the first time in the history of this nation, to endow agriculture in connection with the tuberculosis offensive, with the hope that the present economic loss to the live stock industry will be stopped at the earliest possible date, and last, but most important, is the saving of thousands of our valuable citizens who die annually of tuberculosis.

In this democratic government of ours we must confide in each other, always be alert to our duty as we see it, and if we know our duty we can be depended upon to give our full quota and especially when it is one of public health.

The health section of the League of Nations took notice of the importance of Malta fever, which we have been taught only recently to look upon as one and the same disease as contagious abortion in cattle.

Relative Importance of Malta Fever

I do not know the death rate in Europe from Malta or undulant fever, but, on the other hand, I am sure it has not cost the toll of human lives in America that tuberculosis has, and until its ravages have been more forcibly brought to our attention, do not let us be sidetracked for a moment in our fight on this great white plague.

No other nation has ever attempted to eradicate a disease so widespread as tuberculosis is in this country. However, we have started the fight and we will carry on until the victory is won.

In many instances owners of live stock should be praised for submitting their herds for a tuberculin test. It took great courage on their part in many instances, and now that we have about 900 clean counties, it is easy to see that we can complete the ask on a nation-wide scale. In my opinion, England will also eradicate tuberculosis in the near future.

In this connection, please permit me to digress for just a moment by calling your attention to a subject which has occupied my mind for sometime, and I hope will not fail to awaken your interest, at least mildly.

Improper Distribution of Veterinarians

We, in Oklahoma, and I believe that similar conditions prevail in most of our southern sister states, are suffering not so much from an insufficient number of veterinary practitioners, as from their improper distribution. While our urban centers and more populous and progressive agricultural districts are fairly well supplied in this respect, we have entire blocks of counties, consisting of hundreds of square miles, without a single graduate veterinarian in them. These communities, remote from our larger cities and quite often sparsely settled, do not offer sufficient pecuniary inducements to attract a live up-to-date veterinarian. The result is that we have large areas in which the live stock industry is dependent for its veterinary service and advice, on ignorant empirics, agricultural extension agents, the agricultural press and such assistance as state and federal live stock sanitary officials are able to give. In addition to this, anyone who cares to observe cannot fail to see that the legitimate field of the country practitioner is being invaded more and more by agencies which are attempting to substitute the veterinarian without having the necessary qualifications for doing so. The principal, and sometimes the only, reason for the popularity of this class of service, of doubtful value, is the fact that it is rendered without direct cost to the one receiving it. If this movement is allowed to spread unhindered, it is not so very difficult to foresee the eventual complete elimination of the veterinary practitioner from the field where he is most indispensable, namely, the agricultural and live stock districts of our country.

The question naturally arises: What can and should be done to check this movement, which is bound to react unfavorably on the veterinary profession and incidentally on one of this nation's greatest sources of wealth, our great live stock industry? To me, only one remedy is an *official county veterinarian* in each and every county which deserves the name of being a live stock county.

I have no delusions regarding some of the opposition and other difficulties confronting a plan such as I am proposing, yet I am convinced that these difficulties are not insurmountable and will eventually be overcome. It is my honest conviction that the accreditation of counties as tuberculosis-free and maintaining them in that status will never be entirely successful without the institution of the county veterinarian. If there were no other reason than the successful prosecution of tuberculosis eradication alone, the expense of maintaining a county veterinarian would be fully justified, but there are other reasons equally important. I believe it is the only logical solution of the problem of assuring competent veterinary supervision of the control and eradication of some of our most important animal diseases, and at the same time placing the veterinary profession on the plane where it properly belongs.

It is my fondest hope that this movement may become nation-wide in scope, for the general good and protection of the live stock industry.

Tribute to Two Great Scientists

About fifty years ago, there entered upon the pathway of human endeavor a scientist in the person of Robert Koch, the discoverer of the bacterium of tuberculosis, and a little later he came forth with a biologic product known to us as tuberculin, which has proven the best diagnostic agent known to modern medicine. My friends, it is right for us in this convention here assembled to do honor to the memory of Robert Koch. At the same time, we should do honor to many modern American investigators, at least one of whom we have the honor to have with us today, in the person of Dr. Theobald Smith. I think it right and proper for us to emphasize the work of these men who have devoted their whole lives for the service of others.

My friends, it is my hope that this conference cannot be silenced or subdued until we complete the task which will serve mankind more advantageously than any other human project, namely, the tuberculosis offensive. It is of the highest importance that

this conference was organized, because I have already told you of some of its fruits and a more glorious harvest is yet to come and let our eternal mandate be nothing short of victory.

It is gratifying to me to be here and have the privilege of appearing on this most excellent program. Little did I dream, when down yonder in Texarkana, only three years ago, that it would be possible to bring together such an array of scientific and technical ability as we have been privileged to hear yesterday and today.

In closing, let me say to the officers of this conference that this meeting is all that could be hoped for and more too. I shall long remember this pilgrimage into the heart of a state where hospitality reigns supreme.

Brown Dog Tick Transmits Anaplasmosis

Anaplasmosis has been transmitted experimentally from infected to healthy animals by means of the brown dog tick, *Rhipicephalus sanguineus*, reports Dr. Charles W. Rees, of the U. S. Department of Agriculture, who is in charge of zoological investigations at the New Iberia Experiment Station, Jeanerette, La. Doctor Rees allowed the ticks to engorge as larvae on an animal known to be affected with anaplasmosis and when the nymphs were placed on susceptible animals the disease was transmitted. The experiments were conducted under closely controlled conditions in a specially built barn. All stalls were inclosed with fine-mesh screen to exclude flying insects and the barn was surrounded with a moat of crude oil to exclude crawling forms of life that might affect the results.

Investigators believe that the cattle-fever tick is partly responsible for spreading anaplasmosis, but since the disease occurs also in tick-free areas, it is evident that other carriers are involved in its spread. It has been shown that under experimental conditions the brown dog tick is capable of transmitting this disease and Doctor Rees is conducting further experimental work with this and other species of ticks. Effective control measures are under investigation.

National Dog Week

The week of October 19-25, 1930, has been designated as National Dog Week, with the general purpose of "Better Dogs and Better Care for Them."

THE EFFECT OF THE BRUCELLA GROUP OF MICRO-ORGANISMS ON CHICKENS

Second Paper

By S. H. McNutt *and* Paul Purwin

Department of Veterinary Investigation

Iowa State College, Ames, Iowa

Dubois,[1] in 1910, observed some flocks of chickens, a part of which were sick and dying. Their blood agglutinated *Brucella melitensis* and since sheep on the same farms were found to be infected with *Br. melitensis*, the author concluded that the birds were dying of Brucella infection. Dubois,[2] two years later, evidently reporting the same outbreak mentioned above, says that eleven chickens were examined and four were infected. This conclusion is apparently based on the agglutination test, since nothing is said of isolation of *Br. melitensis*. Mohler,[3] in 1912, reported that chickens had been shown to be infected in one instance when fed the Bang organism. Zwick and Zeller[4] reported negative results in attempts to infect chickens artificially. Koegel[5] showed that feeding and injecting the Brucella produced agglutinins in the blood of chickens. Emmel and Huddleson[6,7] recently reported finding four infected flocks where the disease caused a considerable death loss and great loss in egg production. They considered it serious in such cases. They also report experiments in which they easily infected chickens, producing a high death loss and describe symptoms and lesions. They thought that in some way a toxin might be produced by the infection which would result in death. Emmel[8] reports a fifth flock in which the infection was found and tells of isolation of Brucella from chickens purchased therefrom. Emmel[9] also reports that the turkey, pheasant, pigeon, duck and goose are all susceptible, with the turkey the most easily infected.

In a previous paper[10] we have given the results of experiments in which we attempted to infect chickens and also the results of agglutination tests of several thousand blood samples. In attempts at artificial infection, 103 chickens and various strains of Brucella were employed. Feeding of chickens produced agglutinins which in most cases disappeared within 41 days and in all

Received for publication, June 16, 1930.

cases showed decrease in titre with time. When chickens were injected, the agglutinins appeared earlier and persisted longer. At the end of nearly six months after injection some of the birds still showed a high agglutination titre although most of them had either lost their agglutinins or had a low titre. The longest time following injection when the organism was recovered from injected birds was 22 days. It was not recovered from fed chickens. Visible symptoms were never produced. Evidence of tissue invasion was found in only one case on autopsy. In this case no symptoms were evident, autopsy being performed at 22 days following injection. One other feeding experiment, concluded since the above, showed much the same results. In this experiment 28 mature hens negative to the agglutination test (with Brucella antigen) were fed mixed strains, and 21 days later 13 (62 per cent) showed an agglutination titre of 1:25 up to 1:200. One month later, only one bird showed any agglutinins in her blood. None showed symptoms.

In our previous report, mentioned above, records of 27 flocks showing results of agglutination tests were given. The percentage of birds reacting in dilution 1:25 or higher ranged from 0 to 11. In these flocks, representing 5350 birds, there was an average of 1.7 per cent reactors, with 13 reacting in dilution 1:100 or higher and none reacting above 1:400. Since that time 42 additional

TABLE I—*Results of agglutination tests in 42 flocks*

FLOCK	BIRDS		AGGLUTINATION DILUTION				PER CENT REACTING	BANG'S DISEASE OTHER LIVE STOCK
	TESTED	NEGATIVE	25	50	100	200		
32	200	197	3				1.5	None
33	168	161	5	2			4.3	
35	170	165	4	1			2.9	None
47	200	197	2		1		1.5	
53	145	136	5	2	2		6.2	
54	78	77		1			1.3	
69	250	232	7	6	5		6.4	
71	100	97	1	1	1		3	
72	36	35		1			2.8	
73	70	67	3				4.3	
16 other flocks	1748	1700	33	13	2		2.7	
16 negative flocks	1698	1698	0	0	0	0	0	Bang's disease present on at least one farm
Totals	4863	4762	63	27	11	0	2.1	

farm flocks have been examined, results of which are shown in table I. The fowls in this chart are grouped under 3 classes; first are 10 individual flocks, to show typical examples of flocks showing reactors; then a group of 16 reacting flocks, and lastly 16 non-reacting flocks. The results of these tests are much the same as in the previous tests. The average of all reactors is 0.4 of 1 per cent higher and agglutination titres are lower. No reactions are above 1:100.

For further study all the high reactors were purchased from as many flocks as possible. This resulted in obtaining 19 chickens from 5 flocks. These birds were healthy in appearance when received and have remained so up to the present time, which covers a period of from 2 to 5 months. Other chickens placed with them as checks are also healthy. These high-reacting birds have been tested from time to time and at the last test a month ago all had lost their agglutinins for Brucella excepting two which reacted in low dilution. Some of the reactions are shown in table II.

TABLE II—*Showing loss of agglutinins for Brucella in high-reacting birds from farm flocks*

CHICKEN	FIRST TEST		TITRE ON		
	DATE	TITRE	2–8–30	2–18–30	3–6–30
7844	2–6–30	1:200		1:100	0
8404	2–6–30	1:50		0	0
1135	1–2–30	1:400	1:200	1:100	0
893	12–31–29	1:200		1:25	0
1101	12–31–29	1:400	1:100	1:100	0
1016	12–31–29	1:100	1:50	1:25	1:25
3600	1–16–30	1:200	1:50	1:25	0
C131	1–16–30	1:100	1:25	0	0

DISCUSSION

Attempts have been made to infect 131 chickens artificially with the Brucella organisms. Death has not resulted from infection in any case. Visible symptoms were not encountered. Since artificial infection failed to produce even symptoms, attempts were made to find the infection in farm flocks for comparative purposes. Examination of 69 farm flocks, containing over 10,000 birds, shows less than 2 per cent that agglutinate Brucella in dilutions of 1:25 or higher, with no flock containing more than 12 per cent of reactors. Study of the high-reacting birds from farm flocks shows that they appear healthy and soon

lose agglutinins from their blood. A study of flocks containing reactors indicates little or no unthriftiness present and none caused by Brucella infection. Chickens placed with high reactors remained healthy. In all ways birds that were artificially fed or were injected with cultures behaved as did reacting birds from farm flocks. It appears that chickens on deficient diets are apt to remain reactors longer than those properly fed.

REFERENCES

[1]Dubois, Charles: Malta fever in the fowl. Revue Vet., lxvii (1910), p. 490.
[2]Dubois, Charles: La fievre de Malta a Franquevaux (Gard) en 1910. Rev. Gen. de Med. Vet., xix (1912), pp. 173-183.
[3]Mohler, John R.: Rpt. Chief B. A. I., U. S. Dept. Agr., 1912, p. 357.
[4]Zwick, W., and Zeller, H.: Arbeiten aus dem Reich, xliii (1913), 1.
[5]Koegel, A.: Beiträge zur abortusforschung. Münchner Tierärzt. Wchnschr., lxxiv (1923), p. 617.
[6]Emmel, M. W., and Huddleson, I. Forest: Abortion disease in the fowl. Jour. A. V. M. A., lxv (1929), n. s. 28 (5), pp. 578-580.
[7]Emmel, M. W., and Huddleson, I. Forest: Brucella disease in the fowl. Jour. A. V. M. A., lxxvi (1930), n. s. 29 (3), pp. 449-452.
[8]Emmel, M. W.: An outbreak of Brucella disease in the fowl. Jour. A. V. M. A., lxxvi (1930), n. s. 29 (4), pp. 564-565.
[9]Emmel, M. W.: A preliminary report on the susceptibility of the turkey, pheasant, pigeon, duck and goose to Brucella disease. Jour. A. V. M. A., lxxvi (1930), n. s. 29 (3), pp. 452-453.
[10]McNutt, S. H., and Purwin, Paul: The effect of the Brucella group of microorganisms on chickens. Jour. A. V. M. A., lxxvii (1930), n. s. 30 (2), pp. 212-217.

Conference with Public Health Service

On August 12, 1930, a conference of medical, dental, pharmaceutical, veterinary and other scientific associations and agencies, with officials of the U. S. Public Health Service, was held at Washington, D. C., for the purpose of considering "the question of (a) the necessity for, and (b) the methods to be used in carrying out the provisions of the Act approved June 14, 1930, relating to the quantities of crude opium, coca leaves, and their salts derivatives and preparations, together with such reserves thereof as are necessary to supply the normal and emergency medicinal and scientific requirements of the United States."

Surgeon General Hugh S. Cumming extended an invitation to the American Veterinary Medical Association to send representatives to the conference and President Ferguson appointed, as our delegates, Dr. John P. Turner, Washington, D. C., chairman of the Committee on Legislation; Dr. Roger S. Amadon, University of Pennsylvania, Philadelphia, Pa., member of the Committee on Proprietary Pharmaceuticals; Dr. H. J. Milks, professor of therapeutics, N. Y. State Veterinary College, Cornell University, Ithaca, N. Y.; and Dr. Oscar V. Brumley, Dean, College of Veterinary Medicine, Ohio State University, Columbus, Ohio.

ALASKAN FUR-FARMING CONDITIONS

By EARL F. GRAVES, *Juneau, Alaska*

*Territorial Veterinarian**

In order to have a complete picture of Alaskan fur-farming conditions let us look back a few years when the fox business in Alaska was booming. As soon as it became known that individuals were raising foxes and securing profitable prices for pelts without the usual difficulties and hardships of the trapper, large numbers of people at once flocked into this new field of agriculture. My observations have shown me that the large majority of those who hastened into fur-farming in the Territory were entirely unsuited for any phase of agriculture and most especially for a type of animal husbandry such as fur-farming. It is to be expected that only a comparative few would succeed in other than a mediocre manner; but, the idea gained ground, and owners of stock at once saw the commercial possibilities and disposed of animals at high prices, which was of course good business. Advertising, both honest and dishonest, played (and still is playing) its important part, and thousands of breeding animals were sold.

Alaska has many islands along its coast-line and, to the many persons hastening to engage in the new industry, it seems to have appeared that, in order to become wealthy in a very short period of time, all one need do was to secure one of these islands and liberate upon it a number of foxes. The hypothesis was that the animals would exist and increase with little or no attention and in a year or two the owners would be able to pelt a large number of animals and the resultant income would then make them independent.

After completing an initial survey of Alaskan fur-farming conditions, I could not express any other opinion than that island fox-ranching, as it is practiced in the average run of cases, is extremely wasteful, grossly inefficient and needlessly extravagant. That those not already familiar with island fox-farming may have an idea of how it was, and is, commonly practiced, a brief description is necessary and will suffice. An island may be leased from the government and the farmer then constructs for himself

Received for publication, July 5, 1930

*Resigned.

what buildings are deemed necessary, and a number of blue foxes liberated upon the island. Besides what the animals might gather from the beaches and woods, they may be fed by the owner or caretaker. The feeding varies, but in most cases consists largely of fish or fish offal secured from canneries, cold-storage houses, or by direct fishing. The fish are fed in different forms—fresh, salted and freshened, dried or smoked. This may be fed alone or in combination with such cereals as rolled wheat, oats, rice, barley, or corn meal, cooked into mush in large iron caldrons or steam cookers, or baked.

Various forms of marine life also play important parts in furnishing a source of feed, as does horse meat and beef. The feed was, and is (for many islands ranches have not yet adopted better methods), then placed in coops, trap-houses or box-traps at intervals of several days. During that period of the year when the fur is considered prime, the box-traps and trap-houses in which the animals have been fed during the year are set and the pelts taken. Breeding stock for the ensuing year is liberated at trapping time. The pelts taken are prepared and sent to market and the rancher then hopes for favorable returns and a good crop next year.

Many Unsuited Persons Attracted to New Industry

This type of fox-farming appeals to certain people. It is an easy and an independent life. I do not mean to infer that it is devoid of hard work, for rowing and boating in inclement and even good weather is hard work, as is the felling of trees, procuring of feed and all the manual labor entailed in running any farming proposition, but I do mean to say that to row around an island and throw some food out on the beach, or place it in feed-houses, does not call for a vast amount of ability or knowledge. It is only natural that there was first attracted to fur-farming a certain human element which constantly moves from one occupation to another, seeking something for little or nothing, and fur-farming appeared so easy.

For example, let me cite just two cases of the many coming to my attention. One is of an old gentleman who had been in the light-house service and after watching a few mink-growers, and reading some propoganda, decided that he too would become a mink-farmer. He figured that if he obtained two pairs and that if they each had four young, and the four young produced four young—and so on *ad infinitum*—in a very short time he would, in his own words, "have all the mink in the world." Strange as

it may seem, he did not stop to consider that it was quite likely that all the other people engaged in mink-growing also intended to have "all the mink in the world" in a short time. So, acting on his reasoning, he quit his good, steady job to enter into a new industry of which he knew almost nothing.

Another, a fox-farming organization, leased an island and turned out foxes to run for several years before "the big clean-up." After several years this company placed an advertisement in a Seattle newspaper for an experienced fox-rancher to come to Alaska and pelt this crop of foxes. A cowboy answered the ad and secured the position, although he had never seen a fox. He told me that he sat in the president's office and the president figured that there were so many animals on the island because there had been a certain number liberated which, if figured at so and so and so, would, in the several years that they had been allowed to run, produce a staggering grand total. Of course when the man arrived, there was no such number of foxes present as had been estimated, but there were one hundred and twenty—rather wonderful, considering everything. The place is now a total ruin and many thousands of dollars were literally thrown away. A similar story may be told about many, many islands. Some Alaskan fur-farming organization were—and some are today—out-and-out premeditated swindles; but the majority are of sincere and honest intentions. Far too many engaging in the growing of fur-bearing animals, or entrusting their money to others for this purpose, do not look below a deceptive surface.

A Veterinarian to the Rescue

When results did not conform with expectations, a few of the more thoughtful began to suspect more depth than had been first sounded and began to inquire into causes and possible causes. Territorial aid was solicited and there was employed a veterinarian to assist the fur-farmers and investigate their problems.

A survey established the fact that natural, sane reasons were behind each fur-farm failure instead of the mysterious fancies held by many persons. I have actually had it given to me as a fact that the reason for one certain fox island's failure was due to the breaking of a mirror—and nothing could shake this man's belief in this old superstition.

All animals, or, better still, all individuals are the result of the survival of the fittest. There can be no denying this fact and it is most certainly true of animals living under natural laws and

conditions. Now, since the foxes on the average island ranch do exist under natural conditions, it must be quite obvious that the only possible crop left for the farmer at pelting time is simply what is left after natural factors, either favorable or unfavorable, have taken their toll. This is a common statement of fox ranchers: "I did not get what foxes I should have gotten." In my opinion the farmer making such a statement is simply trying to place the blame at the door of some unknown ill luck instead of on his own shoulders. What might truthfully and correctly have been said is: "I did not get what I *could* have gotten."

Many islands appear to do well for a few years and their sudden misfortune can be traced to sane, logical sources of failures which are but natural unfavorable factors. After the establishment of these causes, all efforts were bent toward an educational campaign to promote better fur-farming methods. An attempt has been made to reach as many fur-farmers as possible and point out these reasons which are now known and proven, and to try to point out practical ways to overcome them.

Parasites Present Problem

There is no great single epizoötic sweeping Alaska. After careful study and much observation, I express the opinion that parasites cause the majority of losses among foxes and that nutritional disturbances cause the greatest losses among mink grown on the fur-farms of Alaska. With foxes, nutritional disturbances rank second, though press forward for first place time and again. Thirdly, the natural tendency of animals to destroy each other is an important factor. Next, follow breeding problems, predatory animals, birds of prey, congenital disturbances, bacterial disturbances and last and least of all is that terrible bugaboo-poaching, which gets the blame for nearly all the other factors. It is easy and comforting for one to accuse his neighbors of theft, but difficult sometimes for some to understand how a number of minute parasites could account for lives. I have examined great numbers of animals and have not found a single case to have been free from parasites at the time of examination. Some cases of parasitism have been pitiful to behold, the hosts being in constant pain and even acute agony. Could anyone reasonably expect such animals to be good parents? Many cases examined have been nearly naked and greatly emaciated from parasitism and malnutrition. Vast numbers of foxes suffer from ear-mite infestation, some cases being malodorous abscesses,

which resulted in the ears being cut off the pelt at skinning time. Lice are a problem on some islands. Internal parasites, especially the round, hook and lung worms, take the largest toll.

Malnutrition is responsible for non-breeding in many cases, abortions, rachitis and a pellagra-like condition. Tail-biting of foxes and mink is the result of malnutrition and is easily corrected by balancing a diet with greens and vegetables and cutting down the amounts of fish or meat feed. There has been an astounding lot of misinformation disseminated concerning the feeding of foxes and mink, and many ranches feed far too much cereal. I have seen hundreds of pounds of feed soured and spoiled because the caretaker did not understand the feeding of his charges. I have seen animals which might have been worth thousands of dollars rendered valueless and hopelessly crippled because of incorrect feeding.

Worthless Nostrums and Fraudulent Advertising

Infections, pneumonias and dystocia have all manifested themselves. Improper medication by incompetent or unwise persons accounts for a large amount of loss. In their efforts to place their various nostrums before the purchasing public, misleading statements are made by houses selling drugs and so-called remedies. One of the most dangerous practices is to attempt to free pregnant vixens of intestinal parasites, yet it is attempted each spring by those who readily believe the advertisements prepared for them.

Many people have claimed that Alaskan foxes are less susceptible to maladies than other foxes. This is a false belief which my findings do not support.

Killings are very numerous among foxes. The males frequently claim more and more area, disposing of other foxes which invade or intrude. Vixens frequently quarrel over puppies or dispose of litters or individuals which can not be cared for. Breeding problems abound and it is conceivable that natural matings are not always the best matings.

I have found many ranchers who turned out for breeding stock animals objectionable or unsuited for propagating purposes, such as males sexually immature, having no testicles descended or but a single one. One man who took steps to investigate conditions on his own ranch found his caretaker about to release two males for breeders which had no testicles.

Birds of prey kill many foxes each year, the eagle being the chief offender, but it is conceivable that the raven may cause more

damage than generally suspected. I have seen mature dogs which had their sight destroyed by ravens picking their eyes, so it may be that some fox puppies meet a similar fate.

There are some areas in Alaska where the mosquitoes are a source of danger—attacking the foxes (and mink) in such numbers as to cause them great pain and constant irritation. It has been found on many occasions necessary to bring animals into the house to protect them against the pests and to medicate the swollen eyes, noses and faces.

Poaching is a potential source of loss but it is my opinion that there is very little thieving done. That it has been practiced has been proven.

The economics of fur-farming are as important as the actual ability to handle the animals. There should be a reasonable amount of good business sense and judgment connected with any enterprise which expects to succeed.

Correct Animal Control

Now, what is the solution of these problems? There can be but one answer in my opinion, and it is *correct animal control*. Not just *any* kind of penning but the *correct* type of penning!

I have been to pen ranches where for three years there had been no increase. The man had never known the sex of his stock. He said: "Oh, I just got twenty foxes and put them in pens. I don't know what they are, but I've never had any increase." Everything about the place was handled with the same degree of inefficiency. This one rancher is not alone. At another place a caretaker told me that it was not discovered for three breeding seasons that all their pairs were females. There are other similar cases. I have seen pen stock suffering from all the unfavorable factors that attack island foxes and even suffering from diseases not likely to attack animals running at liberty. The point I wish to make is that pen ranching can be quite as unprofitable as island ranching; in truth, I know of islands much more profitable than many pen farms, but the fact remains that correct animal control is the only consistently profitable manner to ranch blue and silver fox.

In traveling about advocating correct animal control, many people point out to me certain conspicuous failures in each community and say that animal control is wrong, unnatural, and does not succeed. These folk do not stop to consider that the failures can all be traced to perfectly natural and reasonable causes.

It is not the purpose of this paper to lay down the principles of fox, fur or mink culture. These things are already established and there are many sources of correct information for all who wish to improve their methods.

There need be no alarm by any persons or government organizations that the islands will be abandoned, for a combination of island and pen ranching can be worked out which may prove profitable to each rancher. It is altogether feasible and practical to breed, whelp, mature and secure excellent fur under correct control methods. However, islands could be utilized for furring or mating ranges. It remains for each individual to work towards the ideal just as far as it is practical for him to do so.

Many of the older school of fur-farmers and others not in sympathy with any phase of agriculture dislike to concede that there is a correct and scientific way to raise and handle foxes. However, successful ranchers must do all they can to eliminate guesswork from their operations and control all phases of their business.

Much Progress Now in Evidence

Now, after almost three years have passed, it is indeed encouraging to see much progress. It was a great satisfaction to me not long ago to have one of the older fur-farmers come, asking me to tell him about fox control. At one time I went to this man's island and offered to help him but he did not even invite me ashore. Recently I have been to his ranch and he had pens under construction and was so eager to talk fox culture that he could think of nothing else. * * * All over the Territory I see signs of progress. A few days ago another elderly gentleman came many miles to get a microscope and begged me to accompany him home to teach him how to use it. Several days were spent at his ranch demonstrating how to prepare fecal samples for examination. He talks of vitamins and balanced rations as though he were a college professor. Yet three years ago he scoffed at animal control and fed his foxes by throwing fish-heads on the beach. His stock are now in pens and he is pleased with his results. Recently I assisted a rancher to install a large food-chopper and a bone-grinder in a new house built especially for properly preparing feed. There is an ever-increasing mail from fur-farmers asking for information and I can notice a vast improvement in their questions—for they know more about what they are trying to do than ever before.

There is no intention on my part to assume credit for all the improvement that is taking place in the fur-farming conditions of Alaska. There were a few ranchers just beginning to practice a few phases of animal control at the time I came to the Territory. Then, too, the trade journals have played an important part in raising the plane of activities. The newer fur-farmers do not all begin as the earlier ones did but some come with the better methods already in mind, or ready to use them once they learn of them, and so gradually but surely the growing of fur-bearing animals in Alaska is assuming more pretentious proportions. To date the fur-farmers of the Territory are not a universally prosperous lot, but by the introduction of the correct methods and more attention to details they are helping themselves into an era of better times.

The mink-ranchers as a rule are doing very well, as would be expected, since their stock is under control and little is left to chance or guess-work. Mink, however, do have disturbances which require warding off, and there is much work to be done in an educational way among the mink-growers. Several of the more interesting cases brought to me for autopsy revealed kidney-stones, gall-stones, and a cystic condition of the prostate gland.

Travel from ranch to ranch has been accomplished by boat, rail, auto or foot, and from a few minutes to a number of days were spent in discussing with the ranchers the field of animal husbandry as it applies to fox, mink and fur culture.

There are no large fur-farms in Alaska. It is my opinion that real fur-farming in Alaska is just beginning and is on the threshold of a reasonable growth.

I have worked constantly for the establishment of a small demonstration and experimental fur-farm, so as to provide a place where fur-farmers might have an opportunity to see or know the results of balanced rations, the value of sanitation and disease control. Such a farm would act as a medium for better methods though it would be expensive to establish and maintain. I believe it should be an activity of the Alaska Agricultural College and financed entirely and under the control of the Territory.

In closing I wish to mention that my observations have led me to believe that women are usually better fur-farmers than men, and that I strongly recommend them to be employed by fur-ranching organizations.

THE BLOOD-SUGAR LEVEL OF THE BOVINE*

By E. A. HEWITT,† *St. Paul, Minn.*

Division of Veterinary Medicine, University of Minnesota

Considerable interest has centered around studies on the blood-sugar level in cows ever since it was first suggested that the cause of milk fever might be associated with the amount of sugar in the blood. A fact of greatest interest is that the sugar content of the blood is not so constant as was at one time thought. Especially does this apply to cattle.

The normal level of the sugar in the blood of man has been quite definitely established as between .08 and .18 per cent. In the rabbit, from .045 to .115. In the rabbit, when the low level is reached, peculiar symptoms manifest themselves—violent convulsive seizures which last a minute or so and recur at frequent intervals, followed by a gradual development of a comatose conditions and a fall in body temperature. The symptoms are regarded as being related to the fall in blood sugar (hypoglycemia), since they can be instantly relieved by restoring sugar to the blood by the administration of a solution of glucose. Similar symptoms recur in man if the level drops below .08 per cent.

Hayden and Fish[1] reported the normal level of blood sugar in different animals to be as follows:

	Mg. per 100 cc of blood
Cow	46.52
Horse	102.9
Dog	82
Chicken	139.1
Goats (male, normal)	59.11
Goats (castrated)	56.69

Hayden and Sholl[2] reported that the normal standard of sugar in the blood is definitely lower in the cow, goat and sheep than in the blood of man or dog. They found, in a herd consisting mostly of milk cows, the blood sugar to be 51.75 milligrams per 100 cc of blood (average of 75 tests on 44 cows).

Widmark and Carlens[3] stated that the blood sugar for the dry cow and heifer is around 80 mg. per 100 cc, and for the milking

*Published with the approval of the Director, as Paper 955, of the Journal Series of the Minnesota Agricultural Experiment Station. Received for publication, July 23, 1930.

†Now at the Veterinary Division, Iowa State College, Ames, Ia.

cow as low as 40 mg., with an average of 60 mg. per 100 cc. Auger[4] confirmed the findings of Widmark and Carlens.

Hayden[5] states that his figures from the use of the Benedict method in the determination of sugar in the blood of the milk cow gave a low average of 41.15 mg. per 100 cc of blood.

Schlotthauer[6] reports that the average blood-sugar content in a group of ten non-lactating cows was 65.45 mg. for each 100 cc of blood; the highest was 70.90 mg. and the lowest 59.85 mg.

In a group of lactating cows yielding from 8.30 pounds of milk daily, the blood sugar averaged 63.59 mg. per 100 cc of blood, with a range from 68.45 to 59.85 mg. In another group of lactating cows, yielding from 45 to 58 pounds of milk daily, the blood sugar averaged 61.19 mg. per 100 cc of blood, with a range from 68.72 to 52.9 mg.

Amadon[7] reports that the normal average sugar content of blood samples secured from thirty-five lactating and non-lactating cows and one bull was 68.5 mg. per 100 cc of blood, with a range of from 40 to 86.9 mg. Maguire[8] stated that the sugar content of the blood normally ranges from 80 to 120 mg. per 100 cc of blood.

From the above investigations it is apparent that the sugar concentration in the blood of cows is subject to quite wide variations, but the general conclusion can be drawn that the sugar level is lower in lactating cows than in non-ruminant domesticated animals.

In order to obtain further data upon the normal sugar content of the blood of cows and also the effect upon the blood sugar by different conditions, determinations were made on different groups of cattle at intervals for varying periods of time. In all cases the determinations were made according to the Folin-Wu method of sugar determination.

Table I gives the results of blood-sugar determination made on four heifers, one bull and one steer, each animal being about 12 months of age.

The average of eight blood-sugar determinations on bull 24 over a period of one month was 71.3 mg. per 100 cc. The average of nine determinations on heifer 41, over a period of 53 days, was 71.7 mg. per 100 cc. The average of 27 determinations on another heifer (42), over a period of seven months, was 113 mg. per 100 cc of blood. The average of 27 determinations on heifer 43, extending for a period slightly longer than seven months, gave an average of 118 mg. per 100 cc of blood.

TABLE I—*Blood-sugar determinations on six bovines, all 12 months of age*

DATE	BULL 24	HEIFER 41	HEIFER 42	HEIFER 43	HEIFER 51	STEER X
Oct. 1	59	52		56		
" 5	72			72		
" 10	73	65	69	80		
" 13	69	66	81			
" 19	72	75	72	81		
" 20	69	74	66	83		
" 25	75	71	75	83		
Nov. 1	82	75	76	85		80
" 8		90	87	82		100
" 22		78	77	90		108
" 30			100	107		128
Dec. 7			120	119		
" 14			125	172		
" 15			137			150
Feb. 1			328	332		
" 7			296	296		
" 19			284	362		
" 20			113	120	266	
March 4			85	84	122	
" 10			72	72	78	
" 18			77	83	80	
" 25			92	100	104	
April 2			83	77	66	
" 7			100	93	95	83
" 14			100	100	115	90
May 5			74	79	80	75
" 12			80	93	92	80
" 19			81	89	87	80
June 9			92	90	93	90
Average	71.3	71.7	113	118	106.5	95

Average of 94 determinations—95.9 mg.

It should be noted that on February 21, the blood sugar increased to the very high level of 328 and 332 mg. per 100 cc respectively. On this day these heifers were noticed to be in heat but were not bred. On February 19, the blood sugar of heifer 43 rose to a new high level of 362, whereas in the case of heifer 42 the figure decreased to 284 mg. per 100 cc, which is still a high blood-sugar level. At this time both of these animals were bred and conceived. The very great increase in these figures at first led us to believe that these results were due to faulty technic, but the next day a third heifer (51) was noticed to be in heat and a determination revealed a high figure for that animal also. This animal was bred on that date. This limited number of observations scarcely justifies the drawing of definite conclusions but would indicate a possibility of an increased blood-sugar level being correlated with estrum in heifers.

Twelve determinations on heifer 51, over a period in excess of three months, gave an average of 106.5 mg. of sugar per 100 cc

TABLE II—*Blood-sugar determinations on three non-lactating cows*

DATE	Cow 62	Cow 81	Cow 83
March 26	83	76	71.4
" 28	100	100	86.9
April 19		111	
May 16	95		
Average	92.6	95.6	79.1

Average of 8 determinations—89.1 mg.

TABLE III—*Blood-sugar determinations on three lactating cows*

DATE	Cow E. 81	Cow 21	Cow 70
Jan. 18	38.4		
" 22	33.3		
Feb. 1	68.9		
" 8	58.8	50	
" 14	41.6		
March 7		51.7	63.5
Average	58.2	50.6	63.5

Average of 8 determinations—57.4 mg.

TABLE IV—*Blood-sugar determination in cow affected with pyelonephritis*

DATE	BLOOD SUGAR (MG.)	URINE EXAMINATION					
		REACTION	SP. GR.	ALBUMIN	SUGAR	NaCl	Cl
Jan. 28	80	Acid	1.043	.063	Negative	1.04	.63
Feb. 1	60.6	Acid	1.018	Positive	"		
Feb. 8	80	Alkaline	1.010	Negative	"		
Feb. 14	50		1.005	"	"	.65	.39
Feb. 28	56.3	Alkaline	1.000	"	"		
Average	65.38						

TABLE V—*Blood-sugar determination in non-lactating heifer used in sweet-clover experiment*

DATE	MG.	COAGULATION TIME OF BLOOD
Oct. 5	72	Normal
Oct. 8	82	Normal
Oct. 13	80	Normal
Oct. 19	74	Normal
Oct. 20	74	Normal
Oct. 25	78	Normal
Nov. 1	82	Delayed—partially coagulated in 2 hrs.
Nov. 3	100	Died—blood did not coagulate

of blood. Eleven determinations on a steer gave an average of 95 mg. per 100 cc of blood. An average of 94 determinations on these five animals gave 95.9 mg. of blood sugar per 100 cc of blood.

Table II shows the results of eight determinations on three non-lactating cows. The average of all of these determinations is 89.1 mg. of sugar per 100 cc of blood.

Table III shows the results of eight determinations on three lactating cows, with an average on the three cows of 57.4 mg. per 100 cc of blood.

Table IV gives the results of five blood-sugar determinations on a cow suffering from pyelonephritis, with an examination of the urine. The variation in this condition does not appear to be significant, inasmuch as the observations were not continued through to a termination of the disease.

Table V gives the blood-sugar determinations on a non-lactating heifer which was being used in a sweet-clover experiment. It is interesting to note that the blood sugar mounted to 100 mg. per 100 cc at the time of death.

Observations were made on a lactating cow that had become paralyzed due to obscure cause, other than milk fever. Blood samples were taken before the animal was destroyed and immediately after. Blood sugar determinations in the antemortem blood gave 57.1 mg. per 100 cc of blood and for the postmortem blood 51.28 mg. per 100 cc of blood.

Discussion and Conclusions

The blood-sugar concentration is decidedly higher in heifers and non-lactating cows than in lactating cows. The results of these investigations would indicate that the blood sugar is higher in young cattle before their first lactation period than in non-lactating cows.

There is some evidence that an extremely high blood-sugar level may be correlated with estrum in heifers.

Variations in the blood sugar of a cow suffering from pyelonephritis did not seem to be specific.

The blood sugar rose in a heifer suffering from sweet-clover poisoning at the time of death.

The sugar in the blood after death was lower than that preceding death in the case of a cow suffering from obscure paralysis.

REFERENCES

[1]Hayden, C. E., and Fish, Pierre A.: The normal blood of some domesticated animals. Corn. Vet., xvii (1928), pp. 197-203.

[2]Hayden, C. E., and Sholl, L. B.: A study of the extractives of the blood of the cow. Rpt. N. Y. State Vet. Coll., 1923-1924, p. 102.

[3]Widmark, E., and Carlens, O.: Ueber die Blutzucker Konzentration bei Kuhen und die einfluss der Lactations intensitat auf dieselbe. Biochem. Zeit., clvi (1925). p. 454.

[4]Auger, L.: Recherches sur la pathogene die la fievre vitulaire. Rev. Gen. de Med. Vet., xxxv (1926), p. 353; Abst. Pierre A. Fish, Corn. Vet., xvii (1927), p. 64.

[5]Hayden, C. E.: Sugar, guanidine and cholesterol in the blood of the cow in milk fever. Corn. Vet., xix (1929), 3, pp. 285-295.

[6]Schlotthauer, Carl F.: Theories on the etiology of milk fever. Experimental investigations. Corn. Vet., xvii (1928), pp. 217-224.

[7]Amadon, R. S.: Experimental study of milk fever. Univ. Pa. Vet. Ext. Quar. Bul., xxviii 1928), 16.

Visitors at the Journal Office

The A. V. M. A. Committee on Education met at the JOURNAL office on June 23. Four members were present: Dr. H. E. Bemis, Philadelphia, Pa., *Chairman;* Dr. N. S. Mayo, North Chicago, Ill.; Dr. C. D. McGilvray, Guelph, Ont.; and Dr. Reuben Hilty, Toledo, Ohio. Dr. T. H. Ferguson, president of the A. V. M. A., also was in attendance.

Other veterinarians who were in Detroit at about the same time and who stopped at the office were: Dr. Louis A. Klein, Philadelphia, Pa.; Dr. Cassius Way, New York, N. Y.; Dr. Jacob Traum, Berkeley, Calif.; and Dr. J. P. Bushong, Los Angeles, Calif.

Dr. Cameron W. Argue, of Columbus, Ohio, and Dr. F. A. Humphreys, of Hull, Quebec, also were recent visitors. Local veterinarians who have called at the office the past month include Drs. Wm. B. Whyte, Joseph E. Zeltzer, David Marks, C. M. Hamilton, E. E. Patterson and John Hoberg.

Prof. Dr. Oskar Seifried, of the Veterinary School, University of Gressen, Gressen, Germany, who is spending the year at the Rockerfeller Institute, Princeton, N. J., called at the JOURNAL office, August 9, on his way to Los Angeles, to attend the A. V. M. A. convention.

Badly Needed

The chief constable of a small town was also an expert veterinary surgeon. One night the phone rang, and the constable's wife answered it.

"Is the constable there?" asked an agitated voice.

"Do you want my husband in his capacity of veterinary surgeon or as chief constable?" inquired the woman rather pompously.

"Both, madam," came the reply. "We can't get our new bulldog to open his mouth, and—there's a burglar in it."

Tit-Bits.

HOG INFLUENZA

By J. S. FULTON, *Saskatoon, Sask.*

Veterinary Pathologist, University of Saskatchewan

An investigation was undertaken with a view to determining the cause of losses among swine on a feeding station in Saskatchewan, where from 1500 to 2000 pigs were kept. At the beginning of the outbreak the losses were small, but later as many as ten to fifteen pigs died daily.

Although the possibility of hog choldera being the cause was not seriously considered, it was decided to eliminate such a possibility. Blood from seven hogs, just dead from the disease, was pooled and passed through a Berkefeld filter. Two healthy pigs inoculated with the filtrate remained healthy. Pooled blood from four pigs was introduced directly into two healthy hogs, both of which remained well. Blood cultures from a number of sick hogs proved sterile.

After eliminating hog cholera and failing to demonstrate the presence of *Pasteurella suiseptica*, it was considered possible the losses were due to the disease described as hog "flu."

Although the mortality was much higher than that mentioned by McBryde, Niles and Moskey,[1] the symptoms and postmortem findings so closely resembled hog flu that it was deemed advisable to repeat the work of McBryde[2] in an endeavor to demonstrate his pleomorphic bacillus.

The technic described by McBryde[2] was followed in getting to the lung. The trachea was seared and cut into, cultures made, and mucus collected. Direct examination of the mucus revealed the presence of a small Gram-positive bacillus. Cultures made from bronchial mucus on serum agar (pH 7.5) showed minute transparent colonies in twenty-four hours. The cultured organism in some instances showed a tendency to coccoid forms, but the

majority appeared identical with those taken directly from the mucus.

After repeated transfers, bacillary forms gave way to cocci in short chains and ovoid forms often appearing in pairs. When these changes took place it was noted that Gram stain was no longer retained.

In serum broth (pH 7.5) growth was heavy, falling to the bottom after forty-eight hours. Gelatin was not liquefied. Slight hemolysis on blood-agar plates was noted. Lactose, glucose and mannite were fermented without gas production. Discrete colonies appeared on serum-agar after twenty-four hours. The organism is non-motile.

A faint growth appeared on serum-agar in thirty-six hours, after a suspension had been heated to sixty degrees for sixty minutes. Sixty degrees for ninety minutes killed the organism.

During the course of the investigation, mucus collected from eight affected animals was mixed with saline and blown up the nostrils of a like number of healthy pigs.

The first animal was showered with cold water, with the idea of lowering resistance, but the other seven did not receive this treatment. In every case typical influenza symptoms developed in five to seven days, temperatures ranging from 105 to 107°F. Three pigs so infected were chloroformed and pure cultures of the pleomorphic bacillus recovered. Two of the remaining pigs died as a result of the disease, while the others recovered.

Mucus from a field case was mixed with saline and passed through a Berkefeld filter of N porosity. The filtrate, which proved sterile, was blown up the nostrils of two young pigs, both of which remained healthy.

A serum-agar culture of the pleomorphic organism from a field case was transferred and incubated for twenty-four hours. After washing off the growth with saline, half of the culture was blown up the nostrils of a pig about three months of age. On the sixth day, the temperature rose to 104.6°F. and to 105.2 on the seventh day. Food was refused, and typical signs of flu developed. A control pig, received from the same source as the one inoculated, remained well.

A second pig, given a subculture of the organism, remained well until the fifth day, when the temperature rose to 106.7°F.; on the sixth day, 104.2 was recorded. Temperature remained slightly above normal until the tenth day after inoculation, when it reached 105.7. The pig was then chloroformed and cultures

made from the lungs, which showed a pure culture of the organism with which the animal had been inoculated.

A third pig, given a subculture as in the previous experiments, developed flu symptoms on the sixth day, temperature being 105.2°F. On the seventh and eighth days a rise to 106 was noted. Temperature began to fall on the ninth day, when the animal was chloroformed. Pure cultures were recovered from the lung.

Altogether five pigs were given part of subcultures as already described and in each case typical symptoms developed with a marked elevation of temperature.

Four of the subcultures used in the previous inoculations, when transferred three times, failed to produce the disease when given to healthy pigs. Original cultures from field cases, when kept on ice for two weeks, failed to produce flu symptoms.

A control was used with each pig inoculated, which in every case remained healthy.

The possibility of some of the field cases being infected with *Pasteurella suiseptica* was kept in mind and that this might be determined a rabbit was inoculated and cultures made with the heart-blood from every field case used in reproducing the disease. Cultures proved sterile and all of the rabbits survived.

The outbreak subsided when the weather moderated and with the advent of spring the trouble was over.

It should be here stated that the lobular type of pneumonia, described by McBryde[2] as the cause of most deaths, was found in only one field case examined. The lungs were markedly congested but free from solidification.

The trachea and bronchi contained considerable mucus, which was sometimes streaked with blood. The lymphatic glands were greatly enlarged and congested.

To determine the percentage of animals affected with the disease was impossible, since pigs were being bought and sold almost daily. Owing to conditions existing on the premises, it was difficult to determine the number of the animals which contracted the disease and recovered, but the mortality was undoubtedly high. Housing conditions were not ideal and when the outbreak was at its height the weather was cold, temperatures ranging from 15 to 30 degrees below zero. Although these unfavorable conditions probably had considerable to do with the high mortality, the organism seemed to be particularly virulent, since so little difficulty was experienced in producing the disease in pigs

experimentally, which were kept in good houses and comparatively warm.

Various vaccines were prepared and used both on experimental and field cases but no success was attained.

SUMMARY

1. The work of McBryde, Niles and Moskey was repeated, with results identical.

2. The disease was produced by cultures of the pleomorphic bacillus suspected by McBryde as the causitive organism of swine flu.

3. The organism readily loses its virulence after repeated transfers or when kept a short time.

REFERENCES

[1]McBryde, C. N., Niles, W. B., and Moskey, H. E.: Investigations on the transmission and etiology of hog flu. Jour. A. V. M. A., lxxiii (1928), n. s. 26 (3), pp. 331-346.

[2]McBryde, C. N.: Some observations on "hog flu" and its seasonal prevalence in Iowa. Jour. A. V. M. A., lxxi (1927), n. s. 24 (3), pp. 368-377.

PSAMMOMA IN A HORSE

By E. E. LEASURE *and* E. J. FRICK, *Manhattan, Kansas*

Division of Veterinary Medicine, Kansas State Agricultural College

The patient (C. R. 256) was a large-framed, smooth-mouthed bay gelding, in poor condition, weighing about 1200 pounds. The owner had traded for him two days before our examination. He called us because the horse had not eaten very much and appeared sleepy.

Physical examination on February 6, 1930, revealed normal mucous membranes, temperature 102° F., respirations and pulse slightly decreased. The animal showed symptoms of general nervous depression. When the fore limbs were crossed, he left them in that abnormal position for ten minutes. He was unable to move properly in circles and, when forced to back, he would stagger and throw his head from side to side.

A diagnosis of central nervous disturbance, probably due to chronic dropsy of the lateral ventricles, was made, and a pound of Epsom salt in four gallons of warm water was administered by means of the stomach-tube. Next morning the horse was dead and examination of the brain cavity revealed the presence of psammoma.

These sand tumors originated from the choroid plexus and developed to such an extent that they presented the following

macroscopic appearance. Each psammoma appeared as a somewhat irregular oval dense mass, 7.5 millimeters long by 3 millimeters in diameter. They were joined at their middle and covered with a rather richly vascular glistening capsule. A yellowish, gritty-like substance was scattered throughout the neoplasm.

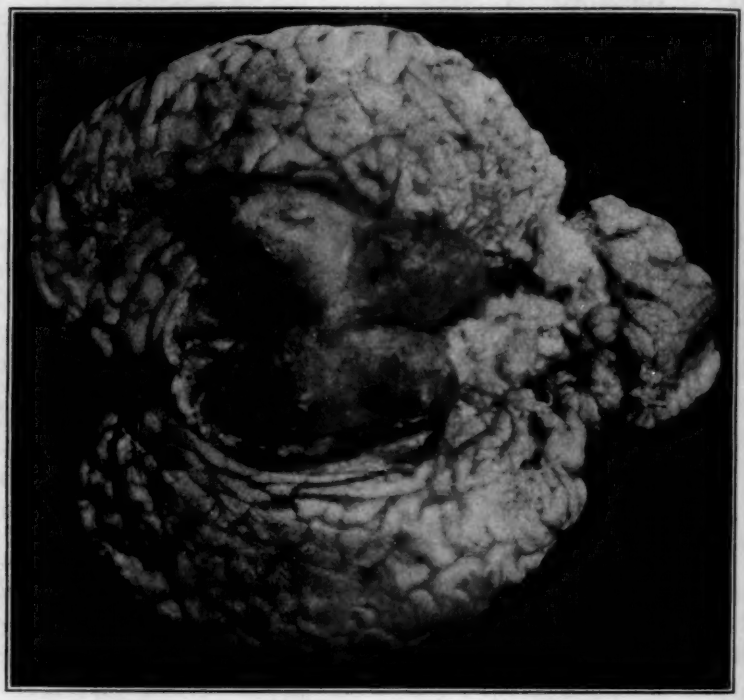

FIG. 1. Psammoma in ventricles of brain of horse.

They occupied the ventricles of the brain. Microscopically they presented a mass of cords and tubes of cells and a conglomerated mass of hyaline spheric masses in which calcium salts had been deposited, the deposits having a microscopical similarity to the particles of so-called "brain sand" (*acervulus cerebri*).

RUBBER HEEL IN STOMACH OF DOG

By E. E. PATTERSON, *Detroit, Michigan*

On April 20, 1930, my assistant, Dr. Kenneth Ross, was called to a local hotel to treat a Scotch Terrier that had been vomiting for a number of weeks. The dog received very little relief from the administration of gastric sedatives which were prescribed.

Five days later the patient was brought to the hospital and given a very careful examination. It was decided to submit the dog to an X-ray examination, which revealed the presence of a foreign body in the stomach.

After consulting with the owner of the dog, it was decided to perform an operation, and the patient was prepared accordingly. My son, Dr. James E. Patterson, was the operator, and the X-ray diagnosis was confirmed by finding one-half of an O'Sullivan rubber heel in the dog's stomach. The patient made an uneventful recovery and five days following the operation was returned

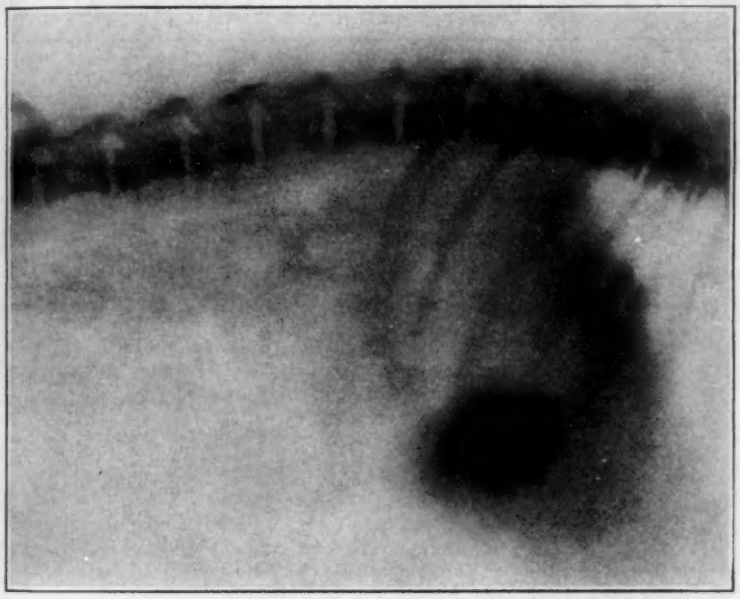

FIG. 1. X-ray photograph showing foreign body in stomach of dog.

to its owner, a prominent actress connected with a musical comedy showing at one of the local theatres.

After the offending rubber heel was removed, the owner of the dog recalled that the patient had been playing with the rubber heel in Chicago, some time previously. She also stated that the dog had been presented to several other veterinarians, in three other cities, for the relief of the gastric disturbance, but the true nature of the cause of the trouble was not suspected. The patient, fully restored to health, and his owner sailed for Panama on June 10. The accompanying X-ray photograph shows the relative position of the rubber heel in the stomach.

SOFT FIBROMA ON THE RIGHT EXTERNAL THORACIC WALL OF A HOLSTEIN COW

By J. F. BULLARD, *Lafayette, Indiana*

Department of Veterinary Science

Purdue University Agricultural Experiment Station

Subject: A grade Holstein cow, with a history of having received an injury on the right chest wall about three years previously. The owner said he treated the cow with "drugs" for a considerable time, which failed to reduce the condition. Clinical examination revealed a large, pendulous growth, 25 inches long, on the

FIG. 1. Lateral view, showing large fibroma.

posterior half of the right thoracic wall. The lower half was much the larger and measured 13 inches from the anterior to the posterior border. The depth was 10 inches. It was attached to the thoracic wall, covering an area six inches wide by eleven inches long. The entire mass was very hard and firm, especially the lower portion.

Treatment: The entire growth was removed under the influence of a local anesthetic, care being taken at all times to control hemorrhage. This was accomplished by ligation. The entire mass peeled out and left a comparatively clean wound. The total weight was 28½ pounds.

Dissection of the growth revealed much dense fibrous tissue in the lower part and considerable gelatinous material, mainly in the upper portions. There were many small kidney- to egg-shaped, firm, fat-like bodies well circumscribed and irregularly scattered throughout the upper part. Microscopic pathology revealed all structures to be connective tissue but of various densities. The small circumscribed bodies were of the loose type, while sections from the lower portions were of a denser variety.

PUNCTURED WOUND

By E. L. KITTRELL, *Augusta, Ark.*

Subject: Bay mare weighing 1500 pounds. The owner called me at night and reported that the mare was sick.

Upon my arrival the patient was lying down. I looked her over and then made her get up. She would not eat, but looked at me as though she thought I was the right fellow at the right time. She seemed to favor the left fore foot.

Treatment: With the aid of my hoof-testers I located a punctured wound which I proceeded to open and treat. This appeared to give the mare relief at once. This case goes to show that all patients, showing what we have come to recognize as colicy symptoms, do not always have colic.

HYPOSPADIA IN A BULL

By MANUEL M. ROBLES, *Manila, P. I.*

Veterinary Research Division, Bureau of Animal Industry

Delafield and Prudden define hypospadia as an arrest of development of the penis and scrotum. They state further:

> In its highest degree the penis is short, the glans penis small. On the lower side of the penis is a deep cleft lined with mucous membrane. Into this cleft the urethra opens at the root of the penis. The scrotum remains separated into two halves, resembling labia majora. The testes may descend into their proper position on each side or remain in the abdomen. If the testicles continue to develop normally the individual has the appearance and capacities of a man; if their development is arrested the individual is apt to be of feminine type.
>
> In lesser grades of hypospadias the two halves of the scrotum are joined and the penis is larger, but a part of the urethra remains open as a cleft at some point of the penis.

The case herewith reported was discovered in a three-year old native bull, one of the hundreds of animals being used annually by the laboratory for the production of rinderpest vaccine. The

unusual seat of urination—the urine passing out spray-like from an opening located at the base of the scrotum, together with the peculiar appearance of the external genital organs of the animal—attracted our attention. On examination of the external genitalia, with a subsequent postmortem inspection of the accessory genital organs, a true type of hypospadia was found.

FIG. 1. (Above.) Hypospadia in a bull, showing the abnormally developed prepuce, penis, and scrotal sacs. The external urethal orifice is over the mark X.

FIG. 2. (Below.) Hypospadia in a bull, showing the normal accessory genital organs and muscles of the prepuce and penis. The external urethral orifice is indicated by a nail over the mark X.

The testicles were well developed but enclosed in separate scrotal sacs, between the bases of which was an opening—the external urethral orifice—5 millimeters in diameter (marked X in the figures). From this opening, a narrow, shallow groove, 5 millimeters in diameter, lined with a thin, hairless, pigmented epithelium, extended anteriorly to the neck of the glans penis. The glans penis was large and knob-like, except for a small blunt

protuberance at its free end, and was adherent to the surrounding skin at its base. The penis was short, the sigmoid flexure being only very slightly flexuous. The prepuce was imperfectly developed, so that the preputial diverticulum as a pouch was absent and instead we found an elongated fossa of the skin covered by an epithelium destitute of hair. Anteriorly, the fossa narrowed to a point where a tuft of hair grew.

The accessory genital organs and the muscles of the prepuce and penis did not show any marked deviations from the normal.

Michigan is Tuberculosis-Free

August 1, 1930, the state of Michigan was officially designated as a modified accredited area, signifying that all its cattle herds are practically free from tuberculosis. This is the third state to attain this distinction. Maine was qualified in March, 1929, and North Carolina in October, 1928.

Final accreditation in Michigan followed closely the recent dismissal of the so-called black cow case by Royal A. Hawley, circuit judge of Ionia, Mich. This case, which questioned the validity of the law authorizing the tuberculin test, was filed against the State by E. S. Townsend, an Ionia County cattle-owner. All other counties in the State had met the official requirements for accreditation. Judge Hawley's decision sustains the right of public authorities to test, condemn, appraise and slaughter privately-owned cattle in the campaign to eradicate bovine tuberculosis. The outcome of the case is therefore considered by veterinary officials to have an important bearing on similar work in other states.

In upholding the State in its successful fight against "one of the most dreaded and deadly diseases that affect humanity," Judge Hawley held that the law is a necessary public-health measure and comes within the authority of the State. "It can not be successfully denied," the decision declared, "that the State has the power to enact legislation requiring the condemnation and destruction of tuberculous cattle and to do so with or without allowing any compensation to the owner for such destruction. The State, however, as well as the federal government, does allow compensation for the animals destroyed."

A few days after the decision was rendered, the cow on which the case rested was slaughtered under the supervision of state and federal veterinarians. The postmortem examination disclosed tuberculous lesions in the lungs and lymph-glands.

PRACTICAL VETERINARY PHARMACOLOGY, MATERIA MEDICA AND THERAPEUTICS. Howard Jay Milks, D. V. M., Professor of Therapeutics and Director of the Small Animal Clinic, New York State Veterinary College, Cornell University. 2nd edition. 539 pages, with 33 figures. Alexander Eger, Chicago, 1930. Cloth, $6.00.

If there should be any doubt about the necessity for a revised edition of this well-known book, all that is necessary to remove such a doubt is a glance at the list of new drugs which have been added to the second edition. Most of these are familiar to veterinarians. The list is too long to enumerate.

The general scheme of the first edition has been retained, although the material has been thoroughly revised and entirely reset. A chapter on biological therapeutics, including diagnostic agents, has been prepared by Dr. Adolph Eichhorn. The chapter on anthelmintics has been carefully brought up to date by Drs. Maurice C. Hall and Willard H. Wright. Names and compositions of official preparations have been made to conform to those of the Tenth Decennial Revision of the U. S. Pharmacopoeia.

The book concludes with an index to prescriptions that are to be found throughout the book. This is a very useful adjunct. In the preface the author very appropriately points out that one cannot practice medicine without prescriptions, and there is an implied note of warning against the present-day tendency of both physicians and veterinarians to depend too much upon ready-made proprietaries.

RABBITS FOR FOOD AND FUR. Frank G. Ashbrook, In Charge, Division of Fur Resources, Bureau of Biological Survey, U. S. Department of Agriculture. 172 pages, with 36 plate illustrations and 8 text illustrations. Orange Judd Publishing Co., Inc., 1930. Cloth, $2.00.

While volumes have been written on the raising of domestic rabbits, it is often difficult for the novice to determine which of the

many conflicting reports should be his guide. The unfortunate exploitation of this growing industry has brought into print many articles containing statements conceived through personal bias. The time is ripe for a publication of this nature by an author with the experience of Mr. Ashbrook whose unique position enables him to judge the merits of various methods without partiality. Every feature involved in the production of domestic rabbits is dealt with in an exhaustive way. For the one who is seeking information on rabbit-raising, this book will serve as a text and may prevent many of the pitfalls.

The problems of feeding, breeding and housing are covered very completely, and sanitation has received special emphasis. It is a recognized fact that many rabbits are lost through unhygienic surroundings and if the suggestions given in this book on hutch construction are followed, it will aid in maintaining the stock in a sanitary condition with a minimum of labor. The information offered is especially practical and is presented in simple and interesting language. The subject matter is divided into 13 chapters covering every feature of this industry, from a general survey of rabbit-raising to methods for preparing the meat for table use.

J. E. S.

PUBLICATIONS RECEIVED

Anthelmintics for the Removal of Thorn-Headed Worms from Swine. Willard H. Wright and H. B. Raffensperger. (Misc. Pub. 79. U. S. Dept. Agr., Washington, D. C., May, 1930.) pp. 11.

Making History with Horses. R. S. Hudson. (Spec. Bul. Michigan State College, East Lansing, Mich., February, 1930.) Illustrated. pp. 48.

Pennsylvania State College Bulletin, General Catalogue, 1930-1931. State College, Pa. pp. 364.

Michigan State College of Agriculture and Applied Science, for the Year 1929-1930, Catalogue of the. East Lansing, Mich., 1930. pp. 343.

The Mink in Captivity. Ronald G. Law. (Bul. 3, Dept. of Game & Fisheries, Toronto, Ont., 1930.) Illustrated. pp. 24.

The Description of a New Fluke Found in the Indian House-Crow (Corvus Splendens). V. R. Phadke. (Bul. 203. Imperial Inst. Agr. Res., Pusa, 1930.) Illustrated. pp. 9.

Kansas Veterinary Medical Association, Report of the Proceedings of the Twenty-Sixth Annual Meeting of the. Manhattan, Kans., January 7-8, 1930. Chas. W. Bower, Secretary, 1128 Kansas Ave., Topeka, Kans. pp. 130.

A Study of the Influence of Bact. Pullorum Infection Upon Some Organic and Inorganic Constituents of the Blood of S. C. White Leghorns. C. E. Hayden and E. L. Brunett. Reprint from *Ann. Rpt.* N. Y. State Vet. Coll., Cornell University, 1928-1929, pp. 135-142.

A Brief Veterinary Survey. Pierre A. Fish. Reprint from *Corn. Vet.*, xx (1930), 2, pp. 101-105.

A Contribution to the Epidemiology of Specific Infectious Cystitis and Pyelonephritis of Cows. F. S. Jones and Ralph B. Little. Jour. Exp. Med., li (1930), 6, p. 909.

The findings indicate that the lower genito-urinary tract of male calves may harbor an organism similar in morphology, cultural characters and certain immunological properties to the organism encountered in this country and Europe in spontaneous infections of the bladder, ureter and pelvic portions of the kidneys of cows. The organism has been found in the sheath, urine and lower portion of the urethra in 12 of 34 calves examined and constitutes but a small proportion of the organisms present. These calves originated in a herd in which infectious cystitis and pyelonephritis existed among the cows. The findings seem to be significant from an epidemiological standpoint. The authors believe that many cases may really originate as infections during early life. The organisms are able to maintain themselves on the mucous membranes of the lower urinary tract where little damage is done but by gradual ascending growth they may finally reach the upper urethra and bladder. When three cows were inoculated intra-urethrally with cultures isolated from the sheaths of calves, two developed transient infections and the other a severe prolonged cystitis and pyelonephritis.

Ovarian Hormone Effects in Ovariectomized Monkeys. D. C. Robertson, W. P. Maddux and E. Allen. Endocrin. xiv (1930), 2, p. 77.

Ovarian hormones were injected into four ovariectomized adult female monkeys. In two, corpora lutea also were implanted. Two experimental menstrual periods followed cessation of injections of estrus-producing ovarian hormone. Sections of the uteri showed typical menstrual endometria of the non-ovulating type. Three implants of recent human corpora lutea did not increase the growth of the endometrial glands beyond the interval stage.

Toward the end of the series of injections it was possible to demonstrate the presence of considerable amounts of glycogen in the epithelial tissue of the uterus and vagina. Glycogen was relatively scarce in the control tissues. Glycogen could also be demonstrated in the epithelial cells sloughed from the vaginal walls. In the vagina it increased in amount as cornification of the epithelial cells progressed. The epithelial lining of the uterine tubes of the injected animals showed remarkable recovery from atrophy following ovariectomy. Mitotic division of cells and regeneration of cilia in some regions were demonstrated. Extensive growth was also induced in the mammary glands including the skin of the nipples. Considerable amounts of the hormone injected were recovered from the urine of experiment animals.

Two New Species of Nodular Worms (Oesophagostomum) Parasitic in the Intestine of Domestic Swine. Benj. Schwartz and Joseph Alicata. Jour. Agr. Res., xl (1930), 6, p. 57.

A morphological description is given of two new species of nodular worms (Oesophagostomum) recently found in the intestines of domestic swine. These species have been namec *O. brevicaudum* and *O. georgianum*, and while the possibility has not been overlooked that the specimens herein assigned to the latter species may represent aberrant forms of *O. dentatum*, it is believed that observed morphological differences warrant the recognition of *O. georgianum* as a distinct species.

Infectious Myxomatosis of Rabbits. Observations on the pathological changes induced by virus myxomatosum (Sanarelli). T. M. Rivers. Jour. Exp. Med., li (1930), 6, p. 965.

The virus of infectious myxomatosis of rabbits (Sanarelli) induces multiple lesions in the skin, lymph-glands, tunica vaginalis, epididymis, testicle, spleen and lungs. Growth and destruction of cells in the epidermis overlying the myxomatous masses leads to the formation of vesicles. Cytoplastic inclusions are seen in other involved epithelial cells. The nature of these inclusions is an open question. In the myxomatous masses situated in the subcutaneous and other tissues, evidences of alteration and growth of certain cells are observed.

THE PRODUCTION AND CURE OF NUTRITIONAL ANEMIA IN SUCKLING PIGS. T. S. Hamilton, G. E. Hunt, H. H. Mitchell and W. E. Carroll. Jour. Agr. Res., xl (1930), 10, p. 927.

A few hours after birth, the hemoglobin concentration in the blood of pigs was found to range from less than 9 to almost 15 gm. per 100 cc, averaging 10.75 in tests on 54 pigs. There is a rapid decrease in hemoglobin during the first few days, a decrease which starts at or soon after birth. In case of litters remaining indoors, this decrease in the level of hemoglobin in the blood continued until concentrations of 4, 3, or even 2 gm. per 100 cc of blood were reached. The birth level of hemoglobin could be restored by administering ferric citrate and copper sulphate to the pigs, either by pipette in daily doses equivalent to 25 mg. of iron and 5 mg. of copper, or by spreading a solution of these metallic salts over the udder of the lactating cow. However, the administration of copper and iron salts to the dam during the last two weeks of gestation and the period of lactation had no appreciable effect on the blood hemoglobin concentration of the suckling pigs. The administration of copper sulphate alone to the suckling pigs was ineffective in promoting regeneration of hemoglobin. Ferric citrate alone, contaminated with a very small amount of copper, was appreciably effective, but much less so than a combination of copper sulphate and ferric citrate.

BIOLOGICAL STUDIES OF THE TUBERCLE BACILLUS. I. Instability of the organism-microbic dissociation. S. A. Petroff and William Steenken, Jr. Jour. Exp. Med., li (1930), 6, p. 831.

The recent advances in the study of other bacteria with application of the dissociation phenomenon have been applied in the study of acid-fast organisms. Rather than to use "R" meaning rough and "S" meaning smooth, the authors prefer to employ the letter "R" to indicate greater resistance and relative avirulence, and "S" to indicate colonies which are more sensitive to environment while possessing for certain species relatively great virulence. "Dissociation" of the avian tubercle bacillus occurred readily on plain gentian-violet-egg medium. The bovine tubercle bacillus is difficult to dissociate, while the human tubercle bacillus is still more difficult to dissociate. Four B. C. G. cultures obtained from various sources were observed to dissociate into two extreme types of colonies. The authors believe that every single bacillus contains two components, "R" and "S."

If the environment is favorable for the development of the "R" component, the offspring will be "R" although the original organism may be "S." Conversely, if the environment is favorable for the "S" and not the "R" component, the "S" will develop.

CAROTENOSIS OF BOVINE LIVERS ASSOCIATED WITH PARENCHYMATOUS DEGENERATION. John S. Buckley, E. C. Joss, G. T. Creech and James F. Couch. Jour. Agr. Res., xl (1930), 11, p. 991.

The results obtained in studies of bovine livers showing carotenosis indicate that the destructive changes found in these livers are, in all probability, due to the presence of a toxic substance, the nature of which has not been determined. Similar destructive changes were induced in the livers of rats which had been fed for a time on affected cattle livers. The typical yellow livers have a characteristic appearance and are usually slightly enlarged with well-rounded borders. The cut surfaces of fresh specimens readily stain instruments and hands a deep yellow. In more advanced cases the livers undergo fibrous changes. Histopathologically, parenchymatous degeneration is the most outstanding change in the earlier stages. In well advanced cases, areas of degeneration and necrosis are extensive, often including whole lobules or groups of lobules. The yellow coloring matter, or pigment, found in the affected livers, has been definitely identified as carotene, and it is thought that the carotenosis in these cases is simply an associated condition in which the excess carotene is stored up in the liver, while other tissues of the body remain unaffected. The authors are of the opinion that the causative agent or toxic substance is a constituent of some plant indigenous to the region or locality where the affected cattle had their origin.

STUDIES ON THE HEMOLYSIN PRODUCED BY ATOXIC STRAINS OF B. TETANI. J. C. Kerrin. Brit. Jour. Exp. Path., xi (1930), 3, p. 153.

Atoxic strains of *B. tetani* secrete as powerful a lysin as do the toxic strains and this lysin, when tested against various substances, behaves in exactly the same manner as the lysin of the toxic strains. Normal serum and cholesterin exert a powerful antagonistic effect against tetanolysin, while lecithin has no antihemolytic power. These actions of serum, cholesterin and

lecithin separate tetanolysin from streptolysin and the hemolysin secreted by *B. welchii*. The author points to the similarity of the lysins to overcome the criticism that "atoxic" strains are not genuine strains of *B. tetani*.

Some Properties of Mucous with Special Reference to its Antibacterial Functions. Neil E. Goldworthy and Howard Florey. Brit. Jour. Exp. Path., xi (1930), 3, p. 192.

Lysozyme was originally described by Fleming as a lytic substance of enzymic nature and found widely distributed in various species of animals and even in vegetables. Cats' tears are entirely devoid of lytic properties when tested against sensitive air bacteria. Saliva usually contains a considerable amount of lysozyme but none could be detected in mucous procured from the stomach or colon, though a small amount can be demonstrated in the saline extracts of dried mucosa. The rabbit manifested very marked lytic powers in both washings and extracts of mucosa, and the colon was much more active than the stomach, the small intestine falling mid-way between the two. Rabbits' saliva was active to a much less degree than colon extract. The guinea pig, *per contra*, reversed this relationship, the stomach being more potent than the intestine. The goat (one) showed a complete absence of lysozyme from its saliva in a dilution of 1:10. Colon washings showed a mere trace of lysis; tears, on the other hand, were quite active. Of all the secretions investigated, saliva appears to contain lysozyme most constantly.

Immunization of Chickens Against Fowl Pox with Dead, Formolized and Phenolized Virus. I. J. Kligler. Brit. Jour. Exp. Path., xi (1930), 1, p. 10.

Fowl-pox virus heated to 56° C. for one hour, or treated with a 0.5 per cent formalin solution for 4 days, no longer produces lesions in susceptible chickens and also fails to induce immunity. Phenolized vaccine (0.25 per cent phenol) produced from the scabs of the lesions still contains active virus 20, 25 and 50 days after its preparation, the survival of the virulence depending on the concentration of the vaccine suspension. One injection of a phenolized vaccine which no longer produces active lesions is sufficient to produce immunity. The failure of heated phenolized vaccine to produce immunity and the success obtained with

the same material unheated indicates that the immunity was induced by the surviving live virus.

A Study of the Relation Between the Time a Cow is Carried in Utero and her Mature Equivalent Butter-Fat Production. J. P. LaMaster and E. C. Elting. Jour. Dairy Sci., xiii (1930), 3, p. 196.

The data indicate that the mature equivalent butter-fat records of Jersey cows are not influenced by the length of time these cows are carried in utero by their dams. The length of time these cows were carried in utero was not influenced by the age of their dams. The average length of the gestation period for 726 Jersey cows was 278.9 days.

Length of Gestations in Jersey Cows. Lynn Copeland. Jour. Dairy Sci., xiii (1930), 3, p. 257.

The average gestation period for the 1075 cases studied was 278.51 days, but there appears to be an equal chance of a cow calving any time between the 271st and the 285th days. The age of the dam apparently has no effect on the length of gestation. Bull calves were carried an average of 1 day longer than heifer calves, 297 bull calves being carried for an average of 277.48 days and the gestations of 282 heifer calves averaging 278.64 days. The author considers the data too scant to draw conclusions relative to twin births, but the results compare with those obtained by other investigators in that the gestations of twins are usually several days shorter than the average. Calves resulting from abnormally long gestations seem to have a better chance of living than do those born prematurely. No correlation could be detected between the length of different gestations in the same individual dam. Cows showing one or more short gestations also exhibited other gestations longer than the average and vice versa.

Irradiated Ergosterol as an Antirachitic for Chicks. F. E. Mussehl and C. W. Ackerson. Poultry Sci., ix (1930), 5, p. 334.

Chicks reared on a basal ration supplemented with two per cent of cod-liver oil made excellent growth with no evidence of rickets. When irradiated ergosterol was added to the same basal

ration in amounts theoretically equivalent to 2, 4, 6, 20 and 50 per cent of cod-liver oil, respectively, growth was subnormal, and the physical appearance of the chicks indicated rickets. The addition of irradiated yeast to the same ration, in amounts theoretically equivalent to 10 and 30 per cent of cod-liver oil, did not result in normal growth.

THE EFFECT OF IRRADIATION ON COBRA VENOM AND ANTIVENIN. Albert Eidinow. Brit. Jour. Exp. Path., xi (1930), 2, p. 65.

The neurotoxin of cobra venom is destroyed only by wave lengths shorter than 2800 A. U. unless sensitized with eosin. The photodynamic action of eosin is arrested by the presence of blood and serum. The active principles of venom and antivenin show different behaviors to irradiation and this in all probability is due to the nature of their chemical structures. Hemolysins and cytolysins can be destroyed by longer wave lengths, *i. e.*, irradiation through vita glass (3300-2800 A. U.). Cobra venom is resistant to heat at 80° to 100° C. and is readily destroyed by irradiation; antivenin is sensitive to heat but resistant to the action of light.

FERRIC CITRATE AS AN INGREDIENT OF MINERAL MIXTURES IN PAIRED FEEDING EXPERIMENTS WITH GROWING SWINE. W. E. Carroll, H. H. Mitchell and G. E. Hunt. Jour. Agr. Res., xl (1930), 10, p. 927.

The administration of ferric citrate at the rate of 3 grams daily to young growing pigs on a ration of yellow corn, soybean oil meal, linseed meal, alfalfa meal, calcium carbonate and sodium chlorid had no effect upon the rate or economy of gains in 12 to 18 weeks of feeding. The paired feeding method was used in this trial, involving 10 paired pigs. In seven pairs of pigs, the blood was examined at the conclusion of the feeding experiment. Among these pairs the pigs receiving iron consistently showed a higher red cell count and a higher iron content of the blood. The authors regard these results as statistically significant but their practical importance as inconsiderable, since the pigs not receiving iron were not anemic. There was no reason to believe that the iron supplement conferred any actual benefits upon the pigs receiving it.

THE GROWTH OF BR. ABORTUS IN SEALED TUBES. G. S. Wilson. Brit. Jour. Exp. Path., xi (1930), 3, p. 157.

Recently isolated strains of the bovine type of *Br. abortus*, though unable to grow aerobically in open tubes, develop satisfactorily in the presence of air if the tubes are suitably sealed. Evidence is brought to show that the growth in sealed tubes is due to the presence of carbon dioxid given off by the seal. It is shown that carbon dioxid is produced by flamed cotton-wool plugs, and to a less degree by melted paraffin, by sealing-wax and by plain red rubber corks. In tubes sealed with paraffined corks, after preliminary flaming of the cotton-wool plug, the carbon dioxid content of the air over the medium is about 1 to 3 per cent.

THE ELECTRICAL CHARGE OF THE VIRUS OF FOWL-POX AND THE AGENT OF THE ROUS SARCOMA. G. M. Findlay. Brit. Jour. Exp. Path., xi (1930), 3, p. 190.

The "blotting paper" method of Bedson and Bland was applied to the virus of fowl-pox and the agent of the Rous sarcoma to determine the electrical charge of each. According to the observations of the author, the virus of fowl-pox is negatively charged at pH 6.6 to 8.0 and the agent of the Rous sarcoma is negatively charged at pH 5.6 to 8.0.

COCCIDIOSIS IN ANOPHELES MOSQUITOES. C. Manalang. Philippine Jour. Sci., xlii (1930), 2, p. 279.

The author reports finding coccidial infection in the mosquito. The oöcysts are found everywhere in the body but most numerous in the thorax and first three abdominal segments. In heavily infected adult mosquitoes the oöcysts are found in the body-cavity, coxae, thorax, adipose tissue, around the brain, and in the labium. The cavity of the mid gut, the brain tissue and the eggs are free from parasites although the latter may be completely and thickly covered with them. In lighter infections the oöcysts are mostly confined to the abdominal cavity. Little is known about the life cycle and developmental forms. Parasitized insects kept in the laboratory at 23° to 25° C. have lived as long as non-infected ones. The author records another case in which coccidial infection was so intense as to cause the death of numerous mosquitoes.

ARMY VETERINARY SERVICE

Regular Army

Major Jacob E. Behney is relieved from further duty at the quartermaster depot, Fort Reno, Oklahoma, and from additional duty as headquarters veterinarian, remount purchasing and breeding headquarters, Fort Reno, Oklahoma, effective on or about July 15, 1930, and assigned to Fort Myer, Va., for duty.

Major Ralph M. Buffington, Fort Myer, Va., will proceed to his home on or about July 20, 1930, to await retirement.

Veterinary Reserve Corps

New Acceptances

Anderson, Walter Raymond	2nd Lt.	R. F. D. No. 3, Worthington, Minn.
Armstrong, David Earle	2nd Lt.	R. F. D. No. 1, West Winfield, N. Y.
Barrett, John Henry	2nd Lt.	R. F. D. No. 6, Norwich, Conn.
Bartlett, Densil Clyde	2nd Lt.	R. R. No. 1, Mount Vernon, Ohio
Brower, Edwin L.	2nd Lt.	822 Poyntz Ave., Manhattan, Kans.
Buck, Robert Maley	2nd Lt.	Howard, S. D.
Caslick, Frederick Geo.	2nd Lt.	R. F. D. No. 27, Newfield, N. Y.
Carter, Erle Rice	2nd Lt.	Boyden, Iowa
DeVries, Theodore M.	2nd Lt.	Orange City, Iowa
Getz, Austin Taylor	2nd Lt.	Edgeley, N. D.
Gochnauer, Oscar Bruce	2nd Lt.	R. R. No. 1, Skyberg, Minn.
Gordon, Joseph Scott	2nd Lt.	Hartington, Nebr.
Guthrie, Richard Sidney	2nd Lt.	R. F. D. No. 6, Greenwich, N. Y.
Hamilton, Lewis Greeley	2nd Lt.	Portland, Kans.
Hinchman, Howard Russell	2nd Lt.	Canal Winchester, Ohio
Hines, George Henry	2nd Lt.	Hume, Ill.
Hopkins, David	2nd Lt.	Madison Ave., W., Winchester, Mass.
Hummon, Ormond John	2nd Lt.	634 E. Defiance St., Leipsic, O.
Jackson, Ralph William	2nd Lt.	1325 E. Jewell Ave., Denver, Colo.
Johnson, Leslie Edwin	2nd Lt.	Hunt St., Melvin, Ill.
Marvin, John Gibson	2nd Lt.	Crescent Place, Almond, N. Y.
Milks, Clifford Howard	2nd Lt.	113 College Ave., Ithaca, N. Y.
Mingle, Carroll Kirkman	2nd Lt.	124 W. 23rd St., Anderson, Ind.
Moore, Earl Neil	2nd Lt.	Warner, Ohio
Moorman, Chauncey Eugene	2nd Lt.	127 Montclair Ave., Pittsburgh, Pa.
Mosbach, Laurence Edward	2nd Lt.	Whittemore, Iowa
Pavey, William Hugh	2nd Lt.	304 S. Howard St., Sabina, Ohio
Robson, Gail Winfred	2nd Lt.	Mount Victory, Ohio
Schantz, Lansing Camp	2nd Lt.	Lowville, N. Y.
Schoffman, Robert John	2nd Lt.	720 3rd Ave. S., Saint James, Minn.
Seagraves, Charles Henry	2nd Lt.	1502 Washington St., Oregon City, Ore.
Sherer, Kenneth B.	2nd Lt.	R. F. D. No. 7, Fremont, Ohio
Skoog, Harry Edwin	2nd Lt.	Nortonville, Kans.
Smiley, Henry DeVore	2nd Lt.	College Hill, Manhattan, Kans.
Stevens, William Vance	2nd Lt.	New Gambier Road, Mount Vernon, O.

Stigers, Arthur Leroy	2nd Lt.	429 9th St., Valley Junction, Ia.
Tyler, Leonard Lewis	2nd Lt.	Hammondsport, N. Y.
Wassenaar, Peter William	2nd Lt.	Maurice, Iowa
Watson, Edgerton Lynn	2nd Lt.	909 Thurston St., Manhattan, Kans.
Wilson, Ralph Austin	2nd Lt.	R. F. D. No. 4, Sabina, Ohio

Promotions

	To:	
Barker, Harry Elmer	Major	10 Dayton Ave., Clinton Springs, N. Y.
Creely, Andrew John	Major	Boise Butcher Co., Boise, Idaho
Dutro, Nicholas	Lt. Col.	1444 Lincoln St., Brownsville, Texas
Hinkle, Truman Blaine	Major	Ashley, Ohio
Kenney, Thomas Gerrard	Captain	Box 689, Toledo, Ore.
Willey, Louis Earl	Major	c/o Sioux Falls Serum Co., Sioux Falls, S. D.
Wooters, Herbert Stanley	Captain	1111 S. Neil St., Champaign, Ill.

Veterinarians at Fort Lewis

Eight veterinarians attended the Reserve Officers' camp at Fort Lewis, Washington, July 6-19, 1930: First Lieut. Walter E. Neary (Colo. '21), Moscow, Idaho; First Lieut. F. M. Bolin (Iowa '29), Corvallis, Oregon; Captain Clarence J. Cook (K. C. V. C. '13), Red Bluff, Calif.; Second Lieut. Marvin R. Hales (Wash. '26), Olympia, Wash.; Lieut. Col. Ora P. Davis (K. C. V. C. '15), Lewiston, Montana; First Lieut. David F. Coyner (O. S. U. '11), Fresno, Calif.; Major Joseph M. Arburua (San Fran. '15), San Francisco, Calif.; and Second Lieut. George D. Loder (McK. '18), Portland, Ore.

First Lieut. E. W. Young, V. C., U. S. Army, is stationed at Fort Lewis.

Short Course in Diseases of Poultry

The College of Veterinary Medicine, Ohio State University, will offer to graduate veterinarians a short course in diseases of poultry, October 20-24, 1930.

The program for each morning will consist of lectures on the more important conditions affecting poultry. The afternoon sessions will be devoted entirely to laboratory work, including demonstrations, dissections, agglutination tests, identification of parasites and parasite ova, and portmortem examinations. On Friday afternoon an interesting and instructive poultry clinic will be held.

The enrollment for this course will necessarily be limited. Application cards will be mailed from the office of Dean Brumley in ample time for registration and registrants will be accepted in the order in which applications are received.

COMMUNICATION

WHY NOT A FIVE- OR SIX-YEAR STRAIGHT VETERINARY COURSE?

To the Editor:

Is there a shortage of veterinarians? I do not believe so. If the government needs men, it can readily obtain them by offering proper and suitable compensation. There is always a demand for gold at the price of brass and if the employers of veterinarians will offer suitable compensation, the best type of men will fight to gain admission to our colleges. The government clearly demonstrated it could get what it wanted. During the World War it wanted ships and it paid the price for men and materials.

Men fight for admission to law, dental and medical schools simply because the leaders of the professions have wisely seen the need of protecting the public and the practitioner, and these schools have repeatedly lengthened their courses and raised their standards, while veterinary medicine, instead of being a five- or six-year straight veterinary course, is still stumbling along in the same old rut with a four-year course. It has made no progress in twelve years.

In New York State, in 1932, nurses will be required to have a four-year academic high-school course, plus two to three years of about eleven months each of hospital training. Some may say that veterinary medicine does not warrant a longer course because of the inadequate compensation. That is just the point.

The more time we put in school, generally speaking, the more we get out of it. Legislators, financiers and economists cannot realize that the veterinarian is worthy of greater recognition because, in their opinion, he is just about on the lowest rung of the professional ladder.

The state, municipality and government are run on a more or less business-like basis. We cannot convince them of our value to the live stock industry if employers of veterinary service can demand and receive men at shameful salaries. This is due chiefly to the fact that the supply exceeds the demand. Too many veterinarians, or an oversupply of any commodity, means that the same can be purchased cheaply. It is getting back to the indisputable law of supply and demand.

The colleges of America should not engage in a spirit of competition in the number of men graduated. The end of the World War found most of the private veterinary colleges closed. The number of horses in cities and on farms has decreased. There was a period which threatened the very existence of some practitioners. The increased value of live stock and the popularity of the dog, ushered in after the war, made things better for the veterinarian. Then like a war hysteria came the cry for more veterinarians. Propaganda was started among the students. It was reported that veterinarians were dying faster than they could be graduated. Most professional men retire when the grim reaper calls them home. As for deaths, what data are available to show that veterinarians were or are dying faster than they are being graduated, and at what rate?

Statistics furnished by the secretary of the A. V. M. A. state that during the years 1923 to 1929, there were 1,081 veterinarians graduated, and deaths of A. V. M. A. members were 308. What percentage of the non-members died? There probably were a great many more. The exact statistics are not available.

While the membership of the A. V. M. A. numbers only from 40 to 45 per cent of the eligible veterinarians, yet we know that the most prominent and successful practitioners are members, and that the A. V. M. A. has done splendid work in organizing and raising the status of the veterinarian. Still much is to be done. We might look to the American Medical Association, which is as stern an any labor union. It has seen the condition into which other professions have fallen and has turned away with a shudder at the thought of what might happen if stern preventive measures were not applied. They have determined that they will fight to maintain higher and higher requirements. They realize that cut-throat competition comes from an oversupply and are determined that physicians will not be placed under too great a handicap in the struggle for existence.

The veterinary colleges of Great Britain are seriously considering lengthening the veterinary course to five years. Good things usually sell themselves. I believe that one reason why so many clamor for admission to other professions is because their leaders have kept up the standards and raised the quality of their graduates.

It is common knowledge that we have not graduated as many men as heretofore, but the question in mind is: "Will there ever

be the demand for turning out veterinarians as there was in the years 1900 to 1915, while the concentration of patients under one roof (small-animal hospitals) continues?"

As good roads and means of transportation improve, cannot the modern veterinarian cover two to three times the territory that the old-timer did? As professional men, can we afford to be placed on an equal basis with trade mechanics and the nursing profession? Are we to go another twelve years without raising our requirements? If law, medicine and dentistry have seen fit to raise their standards, is it not about time that we wake up and make veterinary medicine a five- or six-year course, or are we to continue to cry for more men and remain at our present level in order that employers may have a selection of veterinarians at shameful salaries?

RAYMOND J. GARBUTT.

New York, N. Y., July 1, 1930.

Moredun Institute Staff Augmented

Dr. Henry Dryerre, M. R. C. S., L. R. C. P., Ph. D., F. R. S. E., who has, during the last ten years, held the joint appointments of Professor of Physiology in the Royal (Dick) Veterinary College, and Lecturer in Physiology at Edinburgh University, has been appointed Physiological Biochemist to the Animal Diseases Research Institute, Moredun, Edinburgh. While Dr. Dryerre will relinquish his University post, he will continue his professorial appointment at the Royal (Dick) Veterinary College.

Dr. Dryerre has been intimately associated with Dr. Russell Greig, the Director of the Moredun Institute, in the elucidation of the problem of milk fever, and he brings to the Moredun Institute not only a large experience in veterinary physiology, but a wide knowledge of technic.

Mr. W. S. Gordon, M. R. C. V. S., has received the appointment of Senior Bacteriologist. Mr. Gordon, for the last four years, has been a research assistant at the Wellcome Research Laboratories, and is well known among south country flockmasters in connection with investigations into lamb dysentery and braxy.

These appointments will mark a distinct accession to the strength of the research staff of the Moredun Institute.

U. S. Civil Service Examination

The United States Civil Service Commission announces an open competitive examination for junior veterinarian to fill vacancies in the Bureau of Animal Industry, Department of Agriculture, for duty in the field. The entrance salary for junior veterinarian is $2,000 a year. Higher-salaried positions are filled through promotion.

Competitors will be rated on theory and practice of veterinary medicine and on veterinary anatomy, physiology and pathology, and meat inspection. The duties are to make antemortem and postmortem inspections of food animals and inspection of food products; the administration of tests for disease; control and eradication of disease; and sanitary inspection of establishments and plants, and related duties as directed.

Applications for this examination must be on file with the Civil Service Commission at Washington, D. C., not later than September 23, 1930.

Full information may be obtained from the United States Civil Service Commission, Washington, D. C., or the secretary of the United States Civil Service Board of Examiners at the post office or customhouse in any city.

CERTIFICATE FROM ADMIRAL BYRD

On the next page is a reproduction of a certificate received by the Cutter Laboratory, of Berkeley, California, from Admiral Richard E. Byrd, upon his return from the Antarctic Expedition recently. All of the Labrador huskies taken on the trip were immunized against distemper with Cutter's Homologous Anti-Canine Distemper Serum and Antigen, which was shipped from Berkeley to New York in a refrigerator car and thence to Canada, in thermos bottles, where it was administered to about one

hundred dogs selected for the expedition by "Scotty" Allen. The success of this precautionary measure was attested by a report received by the Cutter Laboratory to the effect that not a single case of distemper developed among the dogs.

CERTIFICATE AWARDED CUTTER LABORATORY

STATE VETERINARY MEDICAL ASSOCIATION OF TEXAS

Registration of seventy-five or more veterinarians was recorded for the twentieth annual meeting of the State Veterinary Medical Association of Texas and the eighth short course for graduate veterinarians, which were held at the A. & M. College of Texas, College Station, June 16-20, 1930.

With Dr. W. Ross Hodges, of Ranger, in the chair, the State Association completed its business sessions Tuesday night, with the election of officers for the new year, resulting as follows: President, Dr. T. O. Booth, Temple; first vice-president, Dr. Frank R. Hecker, Houston; second vice-president, Dr. W. T. Haynes, Farmersville; secretary-treasurer, Dr. D. Pearce, Leonard.

Speakers who honored the Tuesday evening session of the meeting were: Dr. L. A. Merillat, of Chicago, who spoke on veterinary medicine, and Dr. N. F. Williams, of Fort Worth, chief veterinarian of the Live Stock Sanitary Commission, whose subject was "Urgency of a Tuberculosis-Free Milk Supply."

At the Wednesday morning session, Dr. Merillat presented the subject of "Periodic Ophthalmia." Dr. H. J. Milks, of Cornell University, covered "Diseases of the Genito-urinary Organs of Small Animals." Dr. Edwin Jungherr, of the Texas Agricultural Experiment Substation 14, Sonora, discussed "The Pathology of Some Important Sheep and Goat Diseases." Dr. J. S. Watson, a practitioner of Mexia, presented "Acetonemia of Cattle." Dr. T. O. Booth, of Temple, also a practitioner, presented "Post-Patrurient Toxemia in Dairy Cows."

At the afternoon session the following subjects were scheduled:

"Hydrotherapy in Cattle and Horses," Dr. D. Pearce, Leonard, and Dr. A. A. Lenert, College Station.

"Surgical Treatment of Phimosis in a Brahma Bull," Dr. A. A. Lenert, College Station.

"Physical Examination for Diseases of Sheep," Dr. Edwin Jungherr, Sonora.

"Surgery of the Eye in Cattle," Dr. L. A. Merillat, Chicago, Ill.
"Epidural Anesthesia in Cattle and Horses," Dr. A. A. Lenert, College Station.
"The Use of Burdizzo Forceps," Dr. V. A. Scott, Stephenville.
"Collection of Blood Samples for Diagnostic Purposes," Dr. J. S. Watson, Mexia, and Dr. R. C. Dunn, College Station.
"The Eradication and Control of Infectious Abortion in a College Herd," Dr. R. C. Dunn, College Station.
"The Use of Harmones in the Treatment of Sterility," Dr. A. A. Lenert, College Station.

The foregoing program was concluded with a symposium on abortion disease of cattle. The Thursday morning and evening program was as follows:

"Vitamins," Dr. P. W. Burns, College Station.
"Dietetic Sore Mouth of Dog," Dr. A. E. Wharton, College Station.
"A Treatment for Black Tongue of Dogs," Dr. T. O. Booth, Temple.
"Diseases of the Skin in Small Animals," Dr. H. J. Milks, Ithaca, N. Y.
"Small-Animal Hospitals, American and European," Dr. L. A. Merillat, Chicago, Ill.

On Monday, the State Board of Veterinary Medical Examiners met for the purpose of examining applicants for a license to practice in Texas. Monday evening, the lawn about Francis Hall was gaily decorated and a barbecue dinner served to the veterinarians and their wives.

D. PEARCE, *Secretary.*

MICHIGAN STATE VETERINARY MEDICAL ASSOCIATION

The forty-eighth annual meeting of the Michigan State Veterinary Medical Association was held at Michigan State College, East Lansing, June 24-25, 1930.

The meeting was opened with a clinic for both large and small animals. Two methods of treating paralysis of the penis in geldings were demonstrated by Dr. T. H. Ferguson, of Lake Geneva, Wis., and Dr. H. E. Bemis, of the University of Pennsylvania, Philadelphia, Pa. An operation for fistula of the withers was demonstrated by Dr. W. F. Guard, of Ohio State University, Dr. Bemis also demonstrated intravenous chloral anesthesia in the horse. His method has great possibilities for the practitioner in the field, as he can give this anesthetic himself, and prior to the operation.

Two large bulls were presented for diagnosis by Dr. Ferguson. One was a case of lameness and the other an abdominal hernia. A colt with tumors in the sinuses of the head also was presented. Destruction of this case was advised.

The clinic for small animals consisted of several spaying and castration operations in both the dog and cat. Several methods of suturing the wounds and removing the ovaries were demonstrated by Drs. E. E. Patterson and L. H. LaFond, of Detroit; A. E. George, of Flint, and J. Y. Veenstra, of Grand Rapids.

Many favorable comments were made concerning the clinic, during the sessions of the meeting which followed, and these were prompted, of course, by the high caliber of the surgeons who conducted the clinic.

The literary part of the program was held in the ball-room of the Union Building. Dr. Frederick G. Novy, Jr., of the Department of Medicine, University of Michigan, spoke on diseases transmissible from animal to man. Dr. T. H. Ferguson gave a talk on the work of the American Veterinary Medical Association. Professor L. P. Doyle, of Purdue University, gave an excellent illustrated lecture on anemia in pigs.

Following this program, the members and their wives were conducted on a tour of the campus, led by Dean Giltner. At six o'clock we gathered in the Union Building for our annual banquet, with Dr. H. Preston Hoskins, of Detroit, as toastmaster. A good time was enjoyed by all.

Wednesday morning the program was opened with an address by Dr. B. J. Killham, of Michigan State College, who explained his extension project on abortion disease control for Michigan. Dr. H. J. Stafseth, of Michigan State College, gave a talk on "The Present Poultry Situation as it Affects the Veterinarian." A spirited discussion followed, led by Dr. E. C. W. Schubel, of Blissfield. The subject of anesthesia was covered in an interesting and practical way by Dr. H. E. Bemis.

In the afternoon Dr. W. F. Guard gave a talk on surgery and practice. He also demonstrated a method for washing out the contents of the rumen in cattle, with the aid of a heavy-wall, large-diameter stomach-tube. The literary program was then brought to a close by Dr. F. E. Stiles, of Battle Creek, who very ably discussed the diseases of sheep as they occur in Michigan.

The ladies in attendance at the meeting got together and formed an auxiliary. The following officers were elected: President, Mrs. E. B. Cavell, Northville; vice-president, Mrs. John A. Schaefer, Bangor; secretary-treasurer, Mrs. Ward Giltner, East Lansing.

Officers of the Michigan State Veterinary Medical Association for the ensuing year were elected as follows: Dr. John A. Schaefer,

Bangor; first vice-president, Dr. E. B. Cavell, Northville; second vice-president, Dr. M. J. Smead, Rochester; third vice-president, Dr. E. T. Hallman, East Lansing; secretary and treasurer, Dr. E. K. Sales, East Lansing. Dr. W. N. Armstrong, the retiring president, was elected a member of the Board of Directors, for a term of six years, succeeding Dr. J. E. Wurm, of Pigeon, whose term expired.

E. K. SALES, *Secretary.*

ILLINOIS STATE VETERINARY MEDICAL ASSOCIATION

The forty-eighth annual meeting of the Illinois State Veterinary Medical Association was held at Bloomington, July 8-9, 1930. The attendance was larger than at any meeting held in several years, and the interest manifested during the entire session was unusual, despite the weather, which made such attention almost distressing.

The essential feature of this meeting was to pave the way for a possible change in the administration of state veterinary activity, so as to bring animal disease control under veterinary direction, as was voted at the meeting of the Association held in December, 1929. Representatives of the State Department of Agriculture, the University of Illinois, the Farm Bureau, the dairy cattle interests and the Illinois State Veterinary Medical Association convened at the call of Mr. Clifford V. Gregory, editor of the *Prairie Farmer*, and formed a committee for the purpose of beginning the work, calculated to improve the Illinois system of animal disease control. Whether this will result in a plan that will place the direction of animal disease control under the immediate charge of those best fitted to administer the service, or whether there will be a continuation of some slight modification of the present plan, depends in a large measure, at present, upon the recommendations of Mr. Gregory's committee. The veterinary representatives on that committee are Doctors H. D. Chamberlain, Belvidere; H. R. Schwarze, East Saint Louis, and W. Lester Hollister, Avon. Veterinarians are greatly outnumbered by the laymen on the committee.

The two-day program consisted of five sessions. The first was a presentation of the animal-disease-control situation, as viewed by different groups, which include the Director of the Illinois State Department of Agriculture and the Superintendent of the

same department, both of whom rank the Chief Veterinarian in animal disease control in Illinois. Also, the manner in which animal diseases are controlled in Pennsylvania was ably presented by the Director of the Pennsylvania Bureau of Animal Industry, Dr. T. E. Munce. Illinois veterinarians who spoke on the subject were Doctors L. A. Merillat, C. C. Hastings, W. H. Welch and Harry Caldwell.

The second session was devoted to poultry diseases, and eight different phases of the subject were considered by as many authorities. The third session was an informal after-dinner program at which Doctors Ashe Lockhart, of Kansas City, Missouri, and D. A. Eastman, of Cedar Rapids, Iowa, spoke. The fourth session was devoted to a study of the bovine mammary gland. Mastitis was presented by Dr. E. A. Schmoker, of Evanston, and discussed by Dr. G. W. Jensen, of Antioch. The fifth session was a symposium on swine disease. Much of this will doubtless appear in complete form in the veterinary journals.

Officers elected for the ensuing year were: Dr. J. H. Krichel, Alexis, president; Dr. L. N. Morin, McLean, vice-president; and Dr. J. V. Lacroix, Evanston, secretary-treasurer. The next meeting will be held at Springfield, when the legislature is in session.

J. V. Lacroix, *Secretary.*

KENTUCKY VETERINARY MEDICAL ASSOCIATION

The annual meeting of the Kentucky Veterinary Medical Association was held at the Brown Hotel, Louisville, July 9-10, 1930. A large and enthusiastic number of veterinarians attended, to make this one of the outstanding meetings in the history of the Association.

The meeting was called to order by Dr. C. E. Palmer, President, of Shelbyville, at 10 a. m., after which the extremely hot weather was scarcely noticed, so interesting and instructive was the program. Several visitors were present and took part in the discussion of the papers. A conference of the federal and state veterinarians, engaged in tuberculosis eradication in Kentucky, was held during the meeting.

Mayor W. B. Harrison delivered the address of welcome, and Dr. D. E. Westmorland, State Veterinarian, made the response. The following program was then presented:

"Diseases of the Ovaries and Uterus," Dr. L. W. Goss, Ohio State University, Columbus, Ohio.
"Cattle Diseases," Dr. C. H. Case, Akron, Ohio.
"Forage Poisoning in Horses," Dr. Frank Hare, Lexington, Ky.
"Sclerostomes in Horses," Dr. Vilo T. Rose, Elkton, Ky.
"Small-Animal Hospital Business," Dr. H. Gieskemeyer, Newport, Ky.
"Small-Animal Practice for the General Practitioner," Dr. A. J. Steiner, Lexington, Ky.

Officers were elected for the ensuing year as follows: President, Dr. F. H. Riester, Buechel; first vice-president, Dr. A. J. Steiner, Lexington; second vice-president, Dr. W. G. Irvin, Richmond; third vice-president, Dr. G. M. Horton, Mount Sterling; secretary and treasurer, Dr. J. R. Stifler, Lebanon.

J. R. STIFLER, *Secretary.*

VIRGINIA STATE VETERINARY MEDICAL ASSOCIATION

The thirty-seventh annual meeting of the Virginia State Veterinary Medical Association was held at Staunton, July 10-11, 1930, and was attended by about 50 per cent of the veterinarians in the State. Among the outstanding features of the meeting was the interesting surgical clinic which was arranged by Doctors S. C. Neff and H. R. Clemmer. Dr. W. F. Guard, of the Ohio State University, who was the chief operator, is to be highly commended both as a surgeon and as a clear lecturer and demonstrator.

Another outstanding feature of the meeting was the attendance at the meeting of the Ladies' Auxiliary, about thirty members being present. This is a young but going organization and adds much to the annual meetings. It was voted to make a substantial contribution to the loan fund of the Women's Auxiliary to the A. V. M. A.

The meeting was marked by good fellowship and harmony throughout. This feature of the meeting alone was worth more than the cost of attending. The banquet held at the end of the first day was said to have been attended by a larger number of the members and their ladies than any other in the history of the Association.

Among those appearing on the program were Doctors H. H. Adair, Herbert Howard, I. D. Wilson, Meade Ferguson and G. C. Faville. The following officers for the ensuing year were elected: President, Dr. J. P. McDonough, Richmond; first vice-president, Dr. W. H. Grubb, Purcellville; second vice-president, Dr. F. F.

Buck, Rural Retreat; secretary, Dr. I. D. Wilson, Blacksburg; and treasurer, Dr. R. E. Brookbank, Richmond.

It was voted to hold the next meeting in Richmond.

I. D. WILSON, *Secretary*

NORTH DAKOTA VETERINARY ASSOCIATION

The twenty-eighth annual meeting of the North Dakota Veterinary Association was held at the Veterinary Building of the Agricultural College at Fargo, July 10-11, 1930, with Dr. L. A. Benson presiding. The program of this successful meeting was occupied with interesting papers and discussions. There was an attendance of about 45 veterinarians from North Dakota with some visitors from nearby points in Minnesota. The following officers were elected for the ensuing year: President, O. D. Foss, Christine; vice-president, E. V. Lagerberg, Tioga; secretary-treasurer, Lee M. Roderick, State College, Fargo.

Dr. T. H. Ferguson, president of the American Veterinary Medical Association, at the opening session, spoke informally of the national organization, its activities for the year, and the benefits derived from membership in it. The remainder of the morning session was occupied by Dr. J. H. Winslow, of LaMoure, with a report of his experience in treating a series of cases of sweet-clover hemorrhage in cattle last winter. A large quantity of defibrinated blood was secured at the packing-house at West Fargo and taken to the farm at a distance of more than one hundred miles. Some of the blood was used with no ill effects after an interval of five days.

The Thursday afternoon program was opened by Dr. C. P. Fitch, of the University of Minnesota. His discussion of "Bang's Disease and How to Control It in Different Herds" was a thorough description of the present status of our knowledge of the problem. The fundamental principles which are involved in the spread of the disease were carefully described. The means of control to be applied with the cooperation of the owner and the supervision of the veterinarian were then presented. Dr. Frank Breed, of the Norden Laboratories, Lincoln, Nebr., gave a timely "Discussion of Various Swine Diseases," which included diseases of the skin and lungs, anthrax and hog cholera. The informal discussion which followed served to amplify further the details for application in the experience in the field.

The morning session of the second day was occupied by a continuation of the swine program and with a discussion of the prob-

lems encountered in sheep practice. Dr. Chas. Murray, of the Iowa State College, presented an excellent paper entitled, "Studies in Enteritis in Swine." It was well illustrated. The investigations at the research laboratory at Ames have added much to our knowledge and understanding of enteritis, which is a common source of serious loss among swine as well as a frequent complication of hog cholera and the preventive treatment thereof. Dr. Hadleigh Marsh, of the Veterinary Research Laboratory at Bozeman, Mont., then gave a concise but very interesting talk on the problems in sheep practice which confront the practitioners. It was entitled, "Sheep Diseases," and was of especial interest because of the increase in the sheep on North Dakota farms during the last few years, with a consequent greater demand for veterinary services.

Dr. T. H. Ferguson concluded the program with "Common Diseases Met in Cattle Practice." His description of handling bloat and choke, his surgical hints and his demonstrations of operative technic on the udder, head and bovine foot maintained the interest to the close of the meeting.

LEE M. RODERICK, *Secretary*

NEVADA STATE VETERINARY ASSOCIATION

The mid-year meeting of the Nevada State Veterinary Association was held at the University of Nevada, Reno, July 16, 1930.

The afternoon was devoted to the demonstration of post-mortem technic on large and small animals, and the collection of material for bacteriological and pathological examination by Drs. L. R. Vawter and K. W. Niemann.

In the evening the following literary program was presented:

"Use of Nomograph Charts for Calculating Balanced Rations," F. B. Headley, Agricultural Experiment Station, University of Nevada, Reno.
"Anaplasmosis of Sheep," Drs. Robert Dill and L. R. Vawter, Reno.
"Progress of Brucellosis Abortion Control in Nevada," Dr. W. B. Earl, Reno.
"Swine Diseases. Round-Table Discussion, Dr. Robert Jay, U. S. Bureau of Animal Industry, Sacramento, California.

Dr. W. B. Earl, of Reno, was selected as the Association's delegate to the Fifth Annual Conference of Accredited Delegates from State, Territorial and Provincial Veterinary Associations, to be held at the Ambassador Hotel, Los Angeles, California, Aug. 25, 1930, and Dr. G. T. Woodward, of Fallon, as an alternate.

EDWARD RECORDS, *Secretary.*

NECROLOGY

PETER J. MEENAN

Dr. Peter J. Meenan, of Fremont, Nebraska, died May 20, 1930, after an illness of several months. He was a graduate of the Kansas State Agricultural College, class of 1909. He is survived by his widow and one son.

THOMAS E. STAFFORD

Dr. Thomas E. Stafford, of Cleveland, Ohio, died June 15, 1930, as the result of a broken neck sustained in a fall down an elevator shaft. Dr. Stafford was a graduate of Ohio State University, class of 1915, and was in the employ of the U. S. Bureau of Animal Industry. He was located at Fredericktown, Ohio, previous to entering the government service.

EMMETT R. HINKLEY

Dr. Emmett R. Hinkley, of Sandusky, Ohio, died at his home, August 1, 1930, in his 63rd year. He had been in failing health for some time.

Born at North Fairfield, Dr. Hinkley attended local schools, the Milan (Ohio) Business College and Chicago Veterinary College. Upon his graduation from the latter institution, in 1891, he located in Sandusky and practiced there until his death.

Dr. Hinkley joined the A. V. M. A. in 1916. He is survived by one brother and one sister.

HORACE E. RICE

Dr. Horace E. Rice, of Little Rock, Arkansas, died August 3, 1930, after an illness dating back over a year. He was a graduate of the Kansas City Veterinary College, class of 1900, and practiced in Little Rock ever since his graduation. He enjoyed a very extensive practice among the owners of race-horses at one time,

as well as among plantation-owners and, during recent years, owners of small animals. He was a member of the Presbyterian Church and a Mason, and is survived by his widow and two daughters.

J. H. B.

GEORGE G. PEARSON

Dr. George G. Pearson, of Washington, D. C., died suddenly, August 9, 1930. Born in Carlisle, England, in 1865, Dr. Pearson secured his veterinary education at the New York College of Veterinary Surgeons. Following his graduation in 1889, he located in Washington and spent his entire professional career there. About fifteen years ago his health failed and he was compelled to relinquish active practice.

Dr. Pearson was greatly esteemed by his fellow practitioners. He was prominent in Masonic circles and was Past Master of Stansburg Lodge No. 24, Brightwood, D. C. He is survived by his widow, one daughter and three grandchildren.

J. P. T.

Our sympathy goes out to Dr. G. P. Rebold, of Grand Island, Nebraska, in the death of his son, George Ferguson Rebold, aged 13, by drowning at the local beach, on July 4, 1930.

PERSONALS

MARRIAGES

Dr. James A. Muffly (U. P. '29), of Canaan, Conn., to Miss Mirian Obold Faust, at Harrisburg, Pa., June 27, 1930.

BIRTHS

To Dr. and Mrs. Lynn C. Palmer, of Kalamazoo, Mich., a daughter, Margaret Ann, July 8, 1930.

To Dr. and Mrs. E. A. Rolston, of Princeton, Iowa, a son, July 21, 1930.

To Dr. and Mrs. C. B. Strain, of Dunkerton, Iowa, a daughter, Patricia Anne, August 1, 1930.

PERSONALS

Dr. G. A. Handley (Chi. '04) has opened an office at 213 S. Columbus St., Lancaster, Ohio.

Dr. H. A. Turner (Ont. '94) has removed from Pleasanton, Calif., to Redwood City, Calif.

Dr. John H. A. Anderson (T. H. '13), of Monticello, Ind., suffered from an attack of influenza during July.

Dr. Walter N. Peterson (Iowa '30) has located at Belle Plaine, Minn., where he will engage in general practice.

Dr. A. T. Bowen (U. P. '30), of Montvale, N. J., is now located at Westboro, Mass. Address: 15 Church St.

Dr. J. A. Campbell (K. C. V. C. '17), who has been in California for some time, has returned to Williamsfield, Ill.

Dr. H. E. Kreidler (U. P. '21), formerly of Trenton, N. J., is now located at Englewood, N. J. Address: 206 Grand Ave.

Dr. Gail W. Robson (O. S. U. '30) has located in Mount Victory, Ohio, where he plans to develop a general practice.

Dr. F. E. Kling (Ind. '11), of Peru, Ind., was slightly hurt when his automobile went into a ditch, the latter part of July.

Dr. H. L. Simpson (McK. '12) has reported a change of address from Los Angeles, Calif., to 2155 Anthony Drive, San Diego, Calif.

Dr. A. T. McIntyre (Ont. '12), of Brown City, Mich., has recovered from his recent illness and operation, and has resumed his practice.

Dr. M. C. Moses (Cin. '10), formerly of New Carlisle, Ohio, has removed to Springfield, Ohio, and opened an office at 805 W. Columbus St.

Dr. Donald M. Snow (U. P. '30), of Philadelphia, Pa., is now located at the Worcester Veterinary Hospital, 252 Franklin St., Worcester, Mass.

Dr. A. L. Brueckner (U. P. '24), who has been with the Pennsylvania Bureau of Animal Industry, for several years, is now located at College Park, Md.

Dr. Ervin E. Slatter (O. S. U. '30) has accepted a position with Drs. Case and Planz, at the Akron Veterinary Hospital, 50 E. Buchtel Ave., Akron, Ohio.

Dr. Frank H. Brown (Ind. '10) has been reelected secretary of the Indiana State Live Stock Sanitary Board, and ex-officio state veterinarian of Indiana.

Dr. M. R. Steffen (Chi. '04) has resigned as editor of the *Allied Veterinarian*, and has left Indianapolis. He is now located at 203 S. Main St., Hartford, Wis.

Dr. D. E. Westmorland (Ind. '04) has been unanimously reelected state veterinarian of Kentucky by the members of the State Live Stock Sanitary Board.

Dr. J. William Watt (Ont. '23), of New Haven, Conn., has removed to Bay Shore, N. Y., where he is now located at Robbins' Small Animal Hospital, 202 E. Main St.

Dr. C. P. Fitch (Corn. '11), of the University of Minnesota, left Saint Paul the latter part of July for a vacation in the East. Dr. Fitch expected to be away for about six weeks.

Dr. Cameron W. Argue (O. S. U. '30) has accepted a position with the American Society for the Prevention of Cruelty to Animals, 24th St. and Ave. A, New York, N. Y.

Dr. E. C. Howe (Colo. '29), formerly of Brighton, Colo., has removed to Fort Lupton, Colo., where he is now associated with Dr. Geo. H. Carr (Colo. 18), formerly of Brighton, Colo.

Dr. A. A. Trotter (Ind. '10), of Columbus, Ind., was compelled to take the Pasteur treatment about the middle of July, following a wound in one hand, inflicted by a cow affected with rabies.

Dr. J. D. Jones (Tex. '26) has resigned his position with the Pennsylvania Bureau of Animal Industry, at Harrisburg, Pa., and has located at Bastrop, La., where he is engaged in general practice.

Dr. Geo. E. Corwin (U. S. C. V. S. '03), formerly of Hartford, Conn., has accepted the position as Assistant Director in the Division of Animal Industry, Department of Conservation, Boston, Mass.

Dr. L. D. Wheeler (Gr. Rap. '18), who formerly practiced at Vicksburg, Mich., before his entry into the automobile business, has returned to veterinary work and opened an office at Stockbridge, Mich.

Dr. R. S. MacKellar (N. Y. C. V. C. '94), of New York City, recently announced the removal of his offices and the Greenwich Village Veterinary Hospital, to new and larger quarters at 329 W. 12th St.

Dr. E. G. Morningstar (Cin. '15), of Saint Paris, Ohio, and Dr. Frank T. Melhorn (Cin. '18), of Quincy, Ohio, have formed a partnership and opened a new small-animal hospital at 113 South Main St., Piqua, Ohio.

Dr. A. Elgas (Ont. '91), of Hartford, Mich., was struck by a bolt of lightning during a heavy electrical storm, at his farm, the early part of August. A cow and a litter of kittens also felt the effects of the shock. All recovered.

Dr. R. E. Smoot (West. '04), of Madisonville, Ky., has been appointed a member of the Kentucky State Board of Veterinary Medical Examiners, succeeding Dr. A. J. Kay (Chi. '18), of Frankfort, whose term of office expired.

Dr. Walter W. Thompson (Mich. '29), who has been located at the Akron Veterinary Hospital, Akron, Ohio, has accepted a position in the Department of Bacteriology and Hygiene, Michigan State College, East Lansing, Mich.

Dr. J. J. Frey (K. S. A. C. '14) is field manager of the Golden State Milk Products Company, with general offices in San Francisco, Calif. Dr. Frey has requested that his mail be addressed to him at 425 Battery St., San Francisco.

Dr. C. R. Walter (K. C. V. C. '08), of Tulsa, Okla., delivered an address on rabies in the dog, at the meeting of the Tulsa Rotary Club, on August 6. Dr. C. Pedrick (K. C. V. C. '18), also of Tulsa, acted as Rotarian of the day.

Dr. Graciano G. Magno (U. Phil. '23) of the Academic High School, Lingayen, P. I., was confined to the Philippine General Hospital, in Manila, from October, 1929, until some time in March, this year, with a very serious illness.

Dr. J. V. Lange (O. S. U. '20), of Mechanicsburg, Ohio, was painfully injured when kicked by a horse he was called to see, in July. Examination of the brain of the horse subsequently indicated rabies. Dr. Lange was taken to Columbus for treatment.

Dr. J. W. Connaway (Chi. '90), of the University of Missouri, accompanied by his wife and daughter, was among the last-minute additions to the list of American veterinarians attending the World Poultry Congress and the International Veterinary Congress in London.

Dr. D. A. Eastman (Iowa '22), of Cedar Rapids, Iowa, recently opened a new small-animal hospital at 901 19th St., Moline, Ill. Dr. Eastman will continue to operate his hospital in Cedar Rapids under the direction of Dr. D. E. Wright (Iowa '27), formerly associated with Dr. J. C. Flynn (K. C. V. C. '10), of Kansas City, Mo.